Annual AVMA Meeting

Denver, Colorado

August 14-18, 1960

Journal

OF THE

AMERICAN VETERINARY MEDICAL ASSOCIATION

Inclusion Bodies Associated with Viral Diseases

AN ILLUSTRATED article describing briefly the characteristics of inclusion bodies found in 29 diseases of animals and man. Page 161

Peritoneopericardial Diaphragmatic Hernia in a Dog

DIAGNOSIS and SURGICAL CORRECTION of this condition is thoroughly described by three university veterinarians. Page 177

Certification of Swine Herds as VPP-Free

A PRACTICAL discussion of a field method of determining presence or absence of virus pig pneumonia in swine. Page 186

Maduromycotic Mycetomas in Animals

THIS REPORT describes a disease that is known to occur in dogs, cats, and horses but is not commonly diagnosed. Page 192

Vol. 137 August 1, 1960 No. 3

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A unique strain of *Erysipelothrix rhusiopathiae*, isolated ofter several years of research, forms the basis of DURAGEN, an improved erysipelas vaccine offered by Corn States Laboratories.

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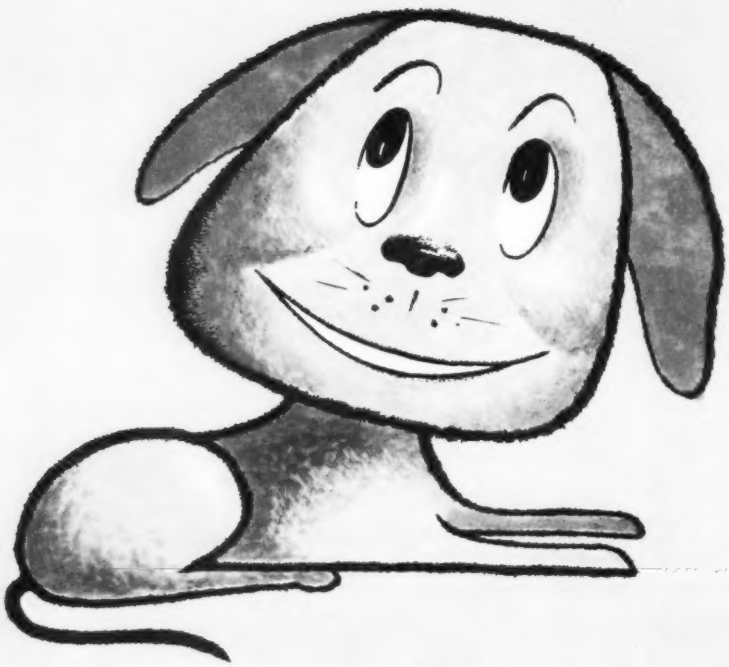

Formula: Each tablet contains Temaril (trimeprazine), 5 mg., added as the tartrate, and prednisolone, 2 mg.

Indications: For the relief of itching and reduction of inflammation in small animal skin disorders such as eczemas caused by internal disorders, fungus infections, otitis, and dermatitis (allergic, parasitic, pustular, and non-specific).

Dosage: Initial dosage is ½ tablet for each 10 lb. bodyweight up to 40 lb.; in patients over 40 lb., 3 tablets twice daily. After 4 days, reduce dosage to ½ of initial dose or as necessary to maintain effect.

In the use of Temaril-P tablets, the desired effect usually is in evidence within 12 hours; other cases may vary from 2 to 72 hours.

Supplied in: Bottle of 100 tablets

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- **Reduces or eliminates need for concomitant therapy**
- **Often relieves itching which does not respond to any other therapy**

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in small animal skin disorders

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Case reports:
"Dog had acute eczema and moist areas over loin from self mutilization, yet did not attempt to scratch or chew at bandage after 48 hours." Virginia.

"The relief is dramatic and is continuing. No noticeable side effects." Florida.

"Owner reports complete remission of symptoms. No recurrence to date." Ohio

"Owner feels the cat is completely changed; no pruritus and excellent coat." California.

"Definitely relieves pruritus." Minnesota.

"The hair is beginning to grow in, and the sheen is returning to the coat. Owners are particularly pleased because this has been a very stubborn case." Florida.

Pruritus therapy: Temaril-P stops the desire to scratch, lick and bite, often relieving pruritus which does not respond to other therapy. It should not, however, be expected to cure pruritus. The cause of the pruritus should be determined and corrected; otherwise, signs are likely to recur following discontinuance of therapy.

Bacterial infections: Temaril-P may be administered to animals with acute or chronic bacterial infections provided the primary cause is controlled by appropriate antibiotic or chemotherapeutic agents.

Precautions and side effects: All the precautions applicable to cortisone or to phenothiazine derivatives apply also to TEMARIL-P. It should be remembered that the premonitory signs of cortisone over-dosage such as sodium retention and edema may not occur with prednisolone. Therefore, the veterinarian must be alert to detect less obvious side effects, such as blood dyscrasias, sedation, polyuria, polydipsia or protruding nictitating membrane which may occur when the dose is excessive. Patients receiving Temaril-P should also be watched for possible occurrence of blood dyscrasias or other serious side effects seen infrequently with some phenothiazine compounds. Less serious side effects, such as sedation and protruding nictitating membrane may occur when excessive dosages are employed. The dosage should then be lowered.

Prolonged treatment with Temaril-P must be withdrawn gradually. If animals under treatment develop excessive thirst, the dosage of Temaril-P should be reduced or discontinued unless the severity of the condition being treated makes its relief paramount.

Do not use Temaril-P in the presence of viral infection (corneal or dendritic ulceration of the cornea).

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Journal

OF THE
AMERICAN VETERINARY
MEDICAL ASSOCIATION

Vol. 137 No. 3 August 1, 1960

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EDITORIAL

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$15.00 per Annum Single Copies, $.75 Prepaid in U.S.
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Correspondence

Comments on Modernization of the J.A.V.M.A.

From some who liked it:

The Penn-Allegheny Veterinary Club, representing forty practitioners from West-Central Pennsylvania, instructed me, its secretary, to write to you to express its satisfaction with the improved format of the JOURNAL. Members were enthusiastic about the features which are concerned with information for the practitioner. They particularly wanted you to know that they feel that the AVMA JOURNAL is now much more useful for them.

s/SAMUEL B. GUSS, Secretary
Penn-Allegheny Veterinary Club
University Park, Pa.

• • •

Ever since having an opportunity to see the new JOURNAL of the AVMA, it has been my intention to write and commend you on the changes that you have made in the publication.

I am indeed glad to see these changes, and I am sure that the AVMA publications will continue to improve and advance.

s/A. H. GROTH
University of Missouri

• • •

Our people here like the new format of the *J.A.V.M.A.*

s/HADLEIGH MARSH
Montana State College

• • •

Congratulations on the new format and other improvements of the JOURNAL as shown by Vol. 136, No. 1 at hand.

More attractive, better grouped into sections, more easily readable print and good pictures.

s/C. E. DECAMP
Scarsdale, N. Y.

• • •

Let me take this means of commending you for the improvement of the 1960 JOURNAL over the ones of the past four or five years. They are much more practical than previously.

s/SIDNEY D. BECKETT
Bruce, Miss.

• • •

May I take this opportunity to offer my congratulations on the dynamic new "face" given the JOURNAL. I am sure that the face-lifting was quite a job for the entire staff.

s/KENNETH B. HAAS
Kalamazoo, Mich.

• • •

I enjoyed the last AVMA JOURNAL. Nice change! Let me know where I can help.

s/JOHN B. HERRICK
Iowa State University

• • •

Congratulations on the fine improvements that were made in the JOURNAL. It is much cleaner looking throughout, making articles more interesting and easier to read.

s/WILLIAM H. FEIGH
Indianapolis, Ind.

• • •

I hope I am among the first to compliment you on the new look imparted to the January 1 issue of the JOURNAL.

I like the new cover, and I consider the new inside layout both more attractive and easier to read.

s/W. W. ARMISTEAD
Michigan State University

Continued on adv. p. 10

CONTENTS—*Continued*

BREAK THROUGH THE EARWAX BARRIER WITH SEBUMSOL™

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□□□ideal new cerumenolytic agent

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□□□without anesthetics or instruments

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news from Washington . . .

FROM THE AVMA WASHINGTON OFFICE
J. A. McCallam, VMD
Brig. Gen. USA (Ret.)

LEGISLATIVE

The following bills have been cleared by Congress for Presidential action:

Surplus Property Transfer — *S. 1018* (in lieu of *H.R. 9600*) authorizes and directs transfer of certain surplus personal property to state and county agencies engaged in cooperative agricultural extension work.

Humane Slaughter — *H. R. 12705,* authorizes 60 days' delay, in limited cases, pertaining to the applicability of certain provisions of law relating to humane slaughter of livestock.

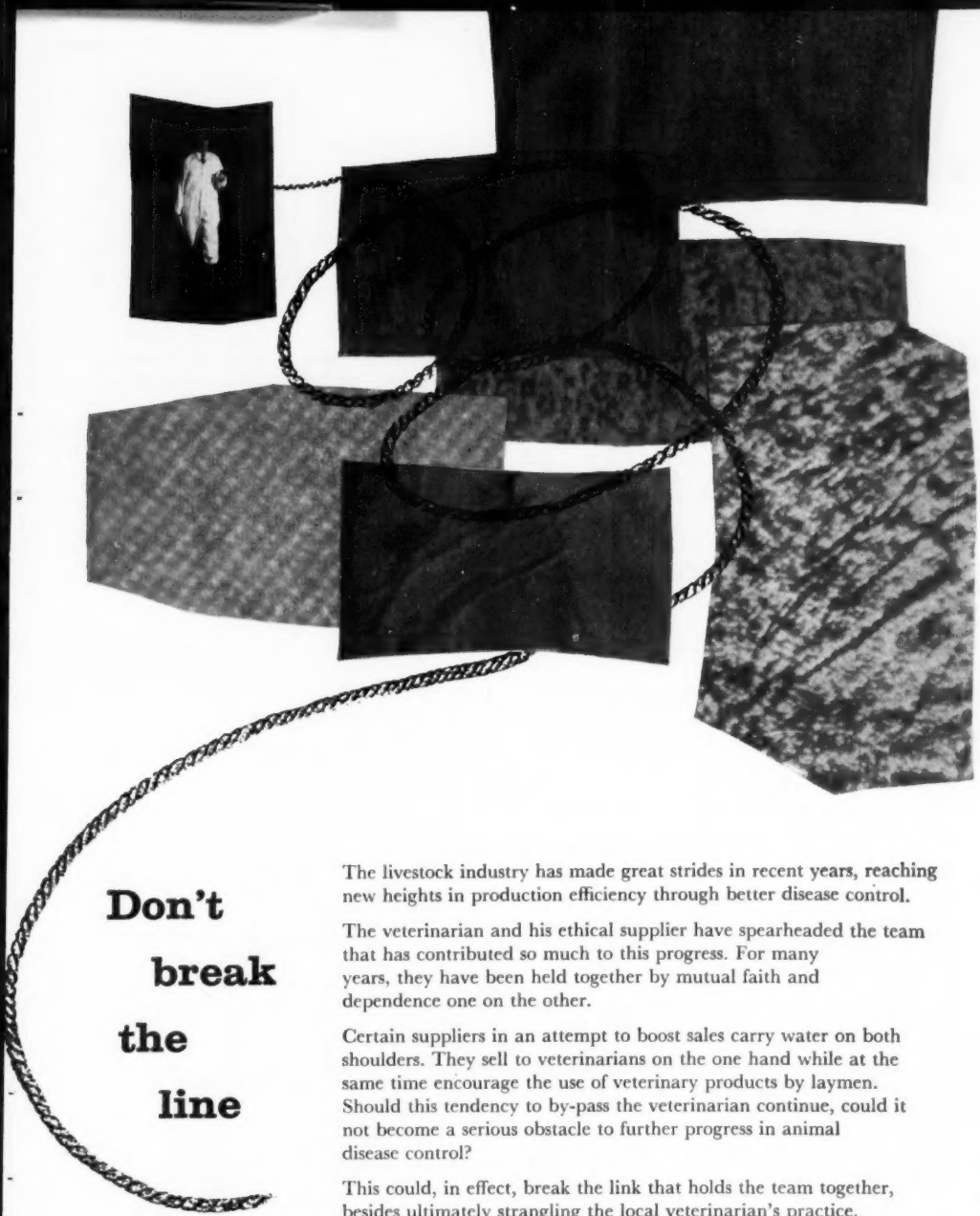
The following bills have been passed by the House:

Color Additives — *H. R. 7624,* color additives, amended. To protect public health by amending Food, Drug, and Cosmetic Act to authorize suitable color additives in accordance with regulations prescribing conditions (including maximum tolerances) under which such additives may be safely used. Bill subsequently vacated and *S. 2197,* similar bill, passed after being amended to contain House-passed language (see JOURNALS May 15, 1960, adv. p. 12; July 15, 1960, adv. p. 14).

International Health Research — *H. J. Res. 649,* International Health Research, passed House June 24, substituting it for *S.J. Res. 41* which had passed Senate (see JOURNAL April 15, 1960, adv. p. 14). Principal differences in House measure—1—does not provide separate institute at NIH, 2—does not specifically authorize new appropriations, rather aims at utilizing foreign currencies and credits, 3—would grant certain powers to the President for purpose of advancing international status of health sciences as distinguished from power granted Surgeon General and Secretary HEW.

Public Health Training — *H. R. 6871.* To amend PHS Act to provide for public health training program. Extension of program expired June 30, 1960. Would authorize new 5-year program of project grants, $2,000,000 annually, in schools of public health; extend without time limit present authority of Surgeon General to make grant-in-aid, not to exceed $1,000,000 annually to public health schools.

Continued on adv. p. 10



Don't break the line

The livestock industry has made great strides in recent years, reaching new heights in production efficiency through better disease control.

The veterinarian and his ethical supplier have spearheaded the team that has contributed so much to this progress. For many years, they have been held together by mutual faith and dependence one on the other.

Certain suppliers in an attempt to boost sales carry water on both shoulders. They sell to veterinarians on the one hand while at the same time encourage the use of veterinary products by laymen. Should this tendency to by-pass the veterinarian continue, could it not become a serious obstacle to further progress in animal disease control?

This could, in effect, break the link that holds the team together, besides ultimately strangling the local veterinarian's practice.

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WASHINGTON NEWS—Continued

Social Security — *H. R. 12580*, Social Security Amendents (see JOURNAL July 15, 1960, adv. p. 16) passed House June 23.

Importing Injurious Animals — *H. R. 10598*, amended, passed House June 24. Clarifies provisions of Criminal Code relative importation injurious mammals, birds, reptiles, etc, and transportation of same (see JOURNAL April 1, 1960, adv. p. 8).

CORRESPONDENCE—Continued

May I congratulate you on the "face-lifting" the JOURNAL has received since the beginning of the year.

I find the JOURNAL easier to read and more pleasant to look at. Keep up the good work.

s/EDWARD BAKER
Englewood, N. J.

• • •

Recent changes in the JOURNAL, in my opinion, have been a fine improvement. Congratulations!

s/F. MURRAY IOBST
Allentown, Pa.

• • •

I have been a member of the American Veterinary Medical Association for approximately 14 years and have enjoyed many past issues of the JOURNAL of the AVMA. It is with this basis for appraisal that I wish to congratulate you on the change which occurred in the *J.A.V.M.A.* in January of this year.

The new cover of the JOURNAL is very attractive, in my opinion, and should do much to promote the profession. In addition, I have gathered a new impression or tone from your manner of presenting the material.

I wish to congratulate you and your staff on improving the *J.A.V.M.A.*

s/E. E. WEDMAN
Arlington, Va.

• • •

What a splendid issue your July 1 is! Just got it this morning.

s/RICHARD D. BURNS
Norwich, N. Y.

• • •

I should like to mention that I like the changes that have been made in the JOURNAL of the AVMA the last few months. In this regard, I feel that the format, style, and appearance of the JOURNAL have been improved greatly. I intended to write a special note about this in January but kept putting it off.

s/W. B. DURRELL
University of Vermont

• • •

The Jan. 1, 1960, *J.A.V.M.A.* issue is very good. Easy to read. The clinicians and researchers should like it. Keep up the good work!

s/GEORGE R. BURCH
New Augusta, Ind.

• • •

I like your new JOURNAL format.

s/D. W. JOLLY
Santa Barbara, Calif.

From the two who didn't:

I received the January 1st issue of the JOURNAL of the AVMA and was quite surprised to see the changes made.

I am very much disappointed with the new cover. It looks like a cheap commercial publication instead of a medical journal. I honestly wish something could be done about this. People that I am in contact with seem to feel the same way.

s/LARRY FREEDMAN
Urbana, Ill.

• • •

I would like to protest the change in the cover of the *J.A.V.M.A.* When the Table of Contents was on the cover, one could obtain an idea of the issue's material by glancing at the cover, without having to thumb through scads of ads to find the index.

The format of the new cover is more becoming to semi-technical journals or Sunday supplements than to the journal of a professional group.

s/LOUIS LOCKE
Laurel, Md.

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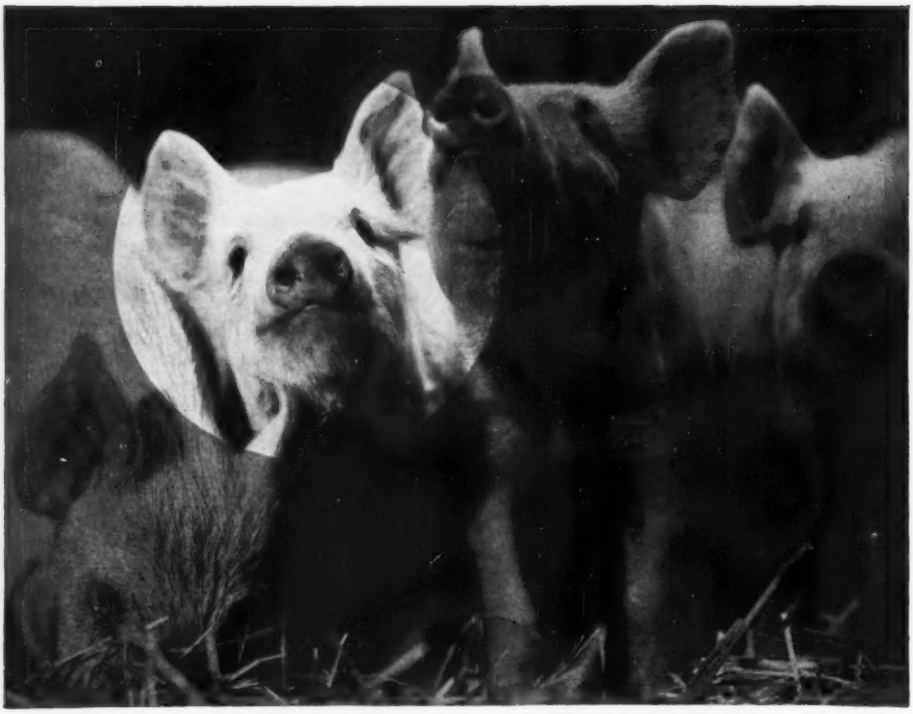

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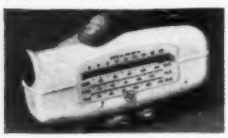

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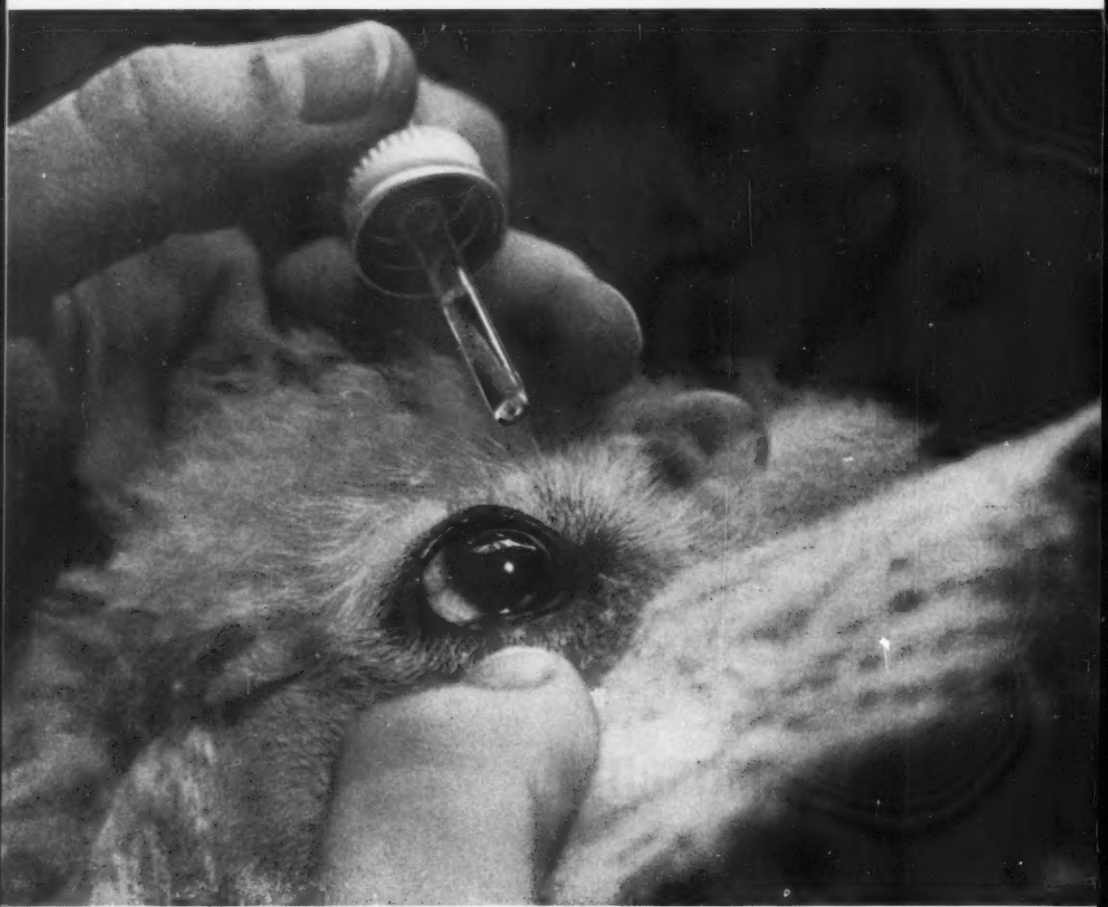

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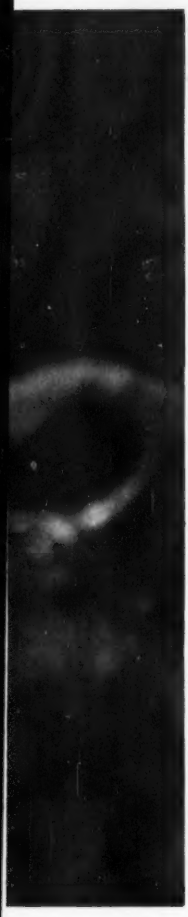

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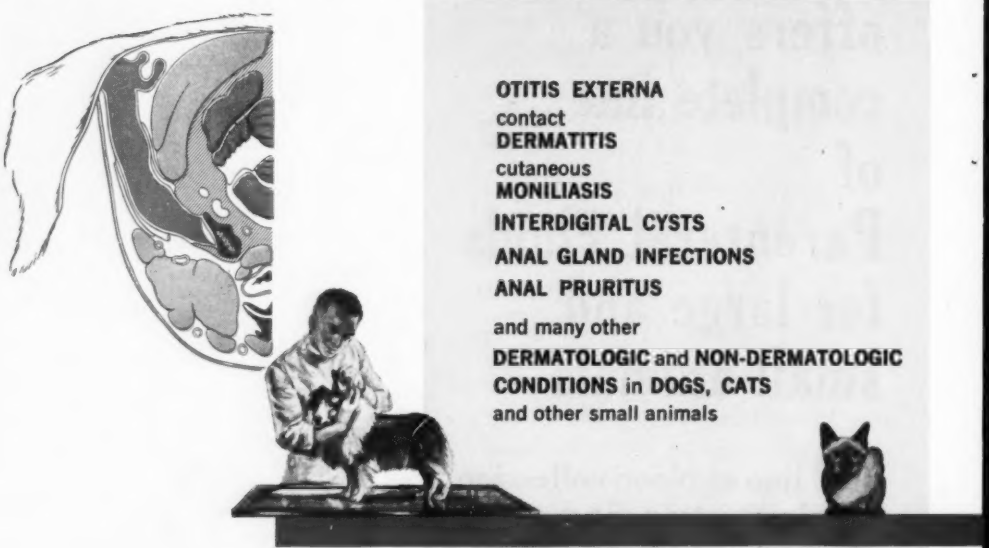

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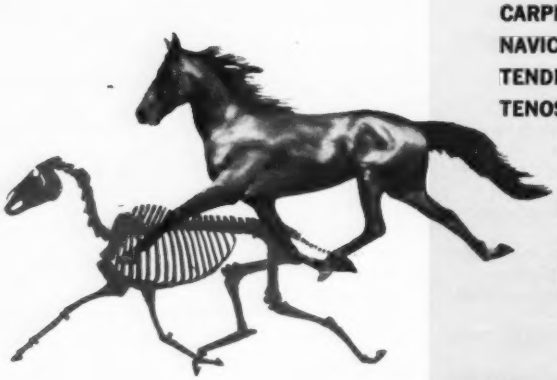

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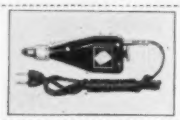

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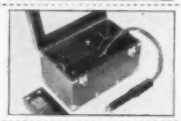

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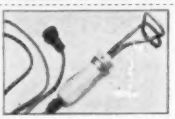

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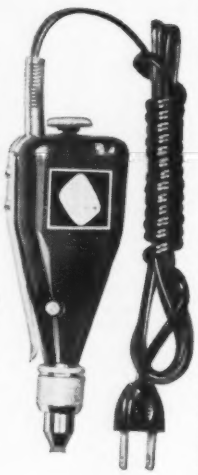
new 10 needle tattoo instrument stops "Collie Nose" symptoms in a hurry



Solar dermatitis, primarily known as "Collie Nose", can be a real problem to treat with lasting results.

Recently, work at the state university here in Colorado showed that tattooing unpigmented areas of the canine's nose with special ink would stop "Collie Nose." Coloring the skin areas stops the irritation of the sun's rays and permits the inflammation to clear promptly and permanently.

Nicholson developed a high-speed electric tattoo instrument with 10 *needles*. This way, whole areas of skin can be tattooed in a hurry and without the usual tedium.

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REFERENCES: 1. Michaelson, S. M., and Covert, M.: J. Am. Vet. M. Ass. 134:334 (Apr.) 1959. 2. Mosier, J. E., and Coles, E. H.: Vet. Med. 53:649 (Dec.) 1958. 3. Belloff, G. B.: Calif. Vet. 9:27 (Sept.-Oct.) 1956. 4. Coles, E. H., and Mosier, J. E.: Am. J. Vet. Res. 20:1020 (Nov.) 1959. 5. Mosier, J. E.: Vet. Med. 52:445 (Sept.) 1957.

Watch for symptoms . . .

Lepto May Come in Disguise

THERE are many "look alike" diseases affecting farm animals. Right now, one which is causing particular concern both to farmers and to livestock health authorities is a complicated problem known as leptospirosis which is causing more than $112 million a year losses in cattle alone.

One of the big difficulties about recognizing and dealing with "lepto" is that it resembles brucellosis in so many ways. In fact, the full extent of the disease in this country has appeared largely as a result ...

In swine, the disease may exist without any apparent symptoms, while in other cases there may be abortions, scouring, loss of appetite—and in severe cases, blood-tinged urine.

As is evident, the chances of confusing leptospirosis with brucellosis are very great, especially with reference to abortions and interrupted breeding schedules. The scouring symptom, also, leads to confusion, as scouring is common in so many other diseases of swine and cattle. For this reason, when ...

in controlling the disease. Thus, if any cattle or swine show symptoms which may indicate brucellosis or "lepto," tests should be made to determine the cause of the condition.

The most important thing to remember is that methods of vaccination have been developed by which leptospirosis can be pre...

Mass Movement Of Stock Always Poses Problem

Annual movement of cattle and sheep from ranges into the farm feedlots of the nation will soon be starting. One of the biggest transportation feats of the year, this ... movement of livestock results in heavy losses caused by shipping fever, according to the American Foundation for Animal ...

Baby Pig Ailments Costing Nearly Half Billion a Year

Anemia, Scours and Transmissible Gastro-Enteritis Are the Problems Which Take the Heaviest Toll —Treatments and Cures Available for First Two.

BABY pig problems are lips, snout and around the ... now costing America's farmers the almost unbelievable sum of nearly a half billion dollars a year. ... eyes, and loss of appetite ... Many times anemic pigs appear thrifty but become tired after even slight movement. Those pigs which do not die probably will never ...

... that the muscles used to expel the gas are inhibited by certain substances. It also is believed by some that "saponins," which are highly concentrated in legumes, affect surface tension. The result is a light, frothy mixture in the rumen which prevents gas from rising readily to the top where it is more easily expelled.

Other theories on the cause of bloat include overfeeding, use of rations to which animals are not accustomed, heredity, soil fertility and climate and weather conditions.

There also is a difference of opinion as to how bloat kills. One theory is that the pressure of the increased volume of gas in the rumen causes suffocation by checking the breathing and flow of blood. Another is that ...

HOG CHOLERA

TAKE only enough hog cholera virus to cover the head of a pin and you can start an outbreak of the most costly single swine disease known in America.

This fact about the nation's No. 1 swine killer assumes special importance for hog producers just now, with the season for heaviest outbreaks of cholera immediately ahead.

"killed" virus vaccines. Each place, but the exact selection matter for the experts.

Authorities have been considerably worried about the hog cholera situation in the past several years ...

Sheepmen Could Stop Ketosis But They Don't

PREGNANCY disease is no respecter of ewes. It attacks fat ewes as well as those in poor condition, and it causes sheep producers an estimated annual loss of animals and wool amounting to more than $1,762,000.

Pregnancy disease is most often ...

One of the main causes of the trouble is the tremendous growth of the unborn lamb during the final stage of pregnancy, thus increasing the ewe's nutritional requirement. If this requirement is not met, the sugar level in the ewe's blood drops, causing ...

Swine Erysipelas Causes $25 Million Annual Loss

Swine erysipelas causes an estimated annual production loss of $25,000,000. It is one of the most widespread and costly of all known diseases of swine.

... diseases with which it appears. This type may be evidenced by the sudden death of one or more hogs before there are any visible symptoms at all.

Acute erysipelas occurs most often in growing pigs. It is the form of the disease which ...

... recent past, and on farms where there is a history of the disease.

Special checks should be made on the health and origin of animals purchased as replacement stock, and such replacement stock should be isolated at least 30 days and ... for possible symptoms ...

veterinary advice

a one-two punch— infertility, sterility

ALMOST EVERY livestock owner is having, or will have, his profits sabotaged this year or next by the "one-two" punch of infertility and sterility.

Cattle are the chief victims of both infertility and sterility. Swine come second, sheep a poor third.

Some estimates figure that a third of all cattle develop some of reproductive problems at one time or another. Other estimates claim that breeding efficiency can be reduced as low as 60 to 80 per cent in many herds.

What are some of the common productive problems, and what can the herd or flock owner do about them? In condensed form, here are a few, what they look like, and what to do:

TRICHOMONIASIS — This disease causes cows to lose their calves. It is transmitted by infected bulls. If a checkup proves the disease is present, the infected bull ... treated and the disease cleared ...

Equally important is a preventive program. In purchasing replacement bulls, their records should be ... and their performance ...

In dealing with swine brucellosis, blood-testing is also a sound starting point. Those pigs which react to the test are marketed, equipment, quarters and premises are disinfected. Then, the remaining pigs are re-tested at intervals and further reactors ...

LAMBING PARALYSIS

PREGNANCY disease is no respecter of ewes. It attacks fat ewes as well as those in poor condition, and it causes our sheep production an estimated annual loss of animals and wool amounting to more than $1,762,000.

The problem is so great that a special effort is ... this month a ...

that feeding is not interrupted, and that the animals have access to water, minerals and salt. The pregnant ewes should also receive plenty of exercise to keep ... liver fat.

America's sheep ... been living with th...

Bloat Adds Up to Heavy Losses

LUSH, FRESH PASTURES, especially legumes, can cause real trouble for stockmen. Annual loss for bloat is now estimated by the American Foundation for Animal Health to exceed $43 million.

The condition of bloat is simple—an excessive accumulation of gas in the rumen, gas which the animal cannot expel. The cause is less certain.

A widespread theory is that legume pastures are risky because the animals do not eat enough roughage to activate ...

LEPTOSPIROSIS

THERE are many "look alike" diseases affecting farm animals. Right now, one which is causing particular concern both to farmers and to livestock health authorities is a complicated problem known as leptospirosis, which is causing more than $112,000,000 a year losses in cattle alone.

One of the big difficulties about recognizing and dealing with "lepto" is that it resembles brucellosis in so many ways. In fact, the full extent of the diseases in this country has appeared largely as a result of our intensified drive against brucellosis. Now, as the drive progresses and widens, we learn that many cases which were thought to be brucellosis are actually its "second cousin," leptospirosis.

Leptospirosis has now been reported in 30 states, and is believed to ...

often affected by a very a... of this disease, which causes scouring in addition to other common symptoms.

In swine, the disease ... without any apparent ... while in other cases there ... abortions, scouring, loss ... —and in severe cases, b... urine.

As is evident, the chances of confusing leptospirosis with ... are very great, especially ... reference to abortions and ... breeding schedules. The ... symptom, also, leads to co... scouring is common in so ... diseases of swine and cattle ... reason, when symptoms ... the first step is to have ... arian identify the exact ... ganism. He may do this ... tory tests of blood, urine, o... sections. Only when the ...

Pneumonia-Enteritis

A DOUBLE-BARRELED disease which attacks young calves, causing scouring and pneumonia complications, is especially prevalent this time of year on Pennsylvania farms. Loss from this disease in the Keystone state is estimated at one and one-half million dollars.

That is the word about its so-called "pneumonia-enteritis complex" issued this month by the American Foundation for Animal ...

Pneumonia-enteritis actually is two diseases — ... and scours. Either disease ... serious, but when a calf ... by both at the same time, ... is greatly increased.

Generally, the disease ... younger calves, from five ... months old, althou...

Watch "Pinkeye"

Most every Wyoming livestock producer is familiar with the age-old problem of "pinkeye", but few realize that it is still costing a total of more than $50,000,00 a year, nation-wide.

In an effort to get something done about these losses, pinkeye has been chosen as a special object of attack as part of National Livestock Loss Prevention Year.

This problem reaches its peak in the summer. It takes its greatest toll among cattle. There are a number of steps which can be ...

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by

ASSOCIATED VETERINARY LABORATORIES

'Pinkeye' Problem Costs Farmers $50 Million Yearly

Alert Farmers To Hog Disease

Don't Relax Defense Against Hog Cholera!

Anaplasmosis Cuts Cattle And Sheep Profits

Internal Parasites Claim Heavy Livestock Toll

WATCH KETOSIS! It Sneaks Up On High Producing Cows

Infertility and Sterility Sabotage Farmers' Profi

Nutritional Diseases Must Be Nipped In Bud

Green Pastures Can Be Big Cause of Livestock Trouble

Bloat, Which Results in 43 Million Dollars Damage Per Year, Is Connected With Grazing Fresh, Lush Legumes—Management Is Only Method of Control.

Fall Is The Time To Watch For . . .

Shipping Fever

Swine Erysipelas

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No. 3
August 1, 1960

Journal
OF THE
AMERICAN VETERINARY
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Inclusion Bodies Associated with

Viral Diseases

of Man and Other Animals

Robert T. HABERMANN, D.V.M., M.S.
Fletcher P. WILLIAMS, Jr., B.S.
George L. FITE, M.D.

THIS ARTICLE presents information on some of the naturally occurring and experimentally induced viral diseases that can be recognized by their effects on the different organs and tissues of laboratory animals and by the formation of characteristic inclusion bodies. The authors, in reporting these diseases, have made no attempt to confirm the reported references, except in instances indicated. (For precise information concerning the fundamental properties of human and animal inclusion bodies and viruses, *see* references 54a and 55a.) The discussion and photography were done by the authors and the National Institutes of Health photographic service.

In addition, information on some of the more important viral diseases of man and other animals, with their pathologic changes in inoculated laboratory animals are tabulated (table 1).

Diseases in Which Inclusion Bodies Occur

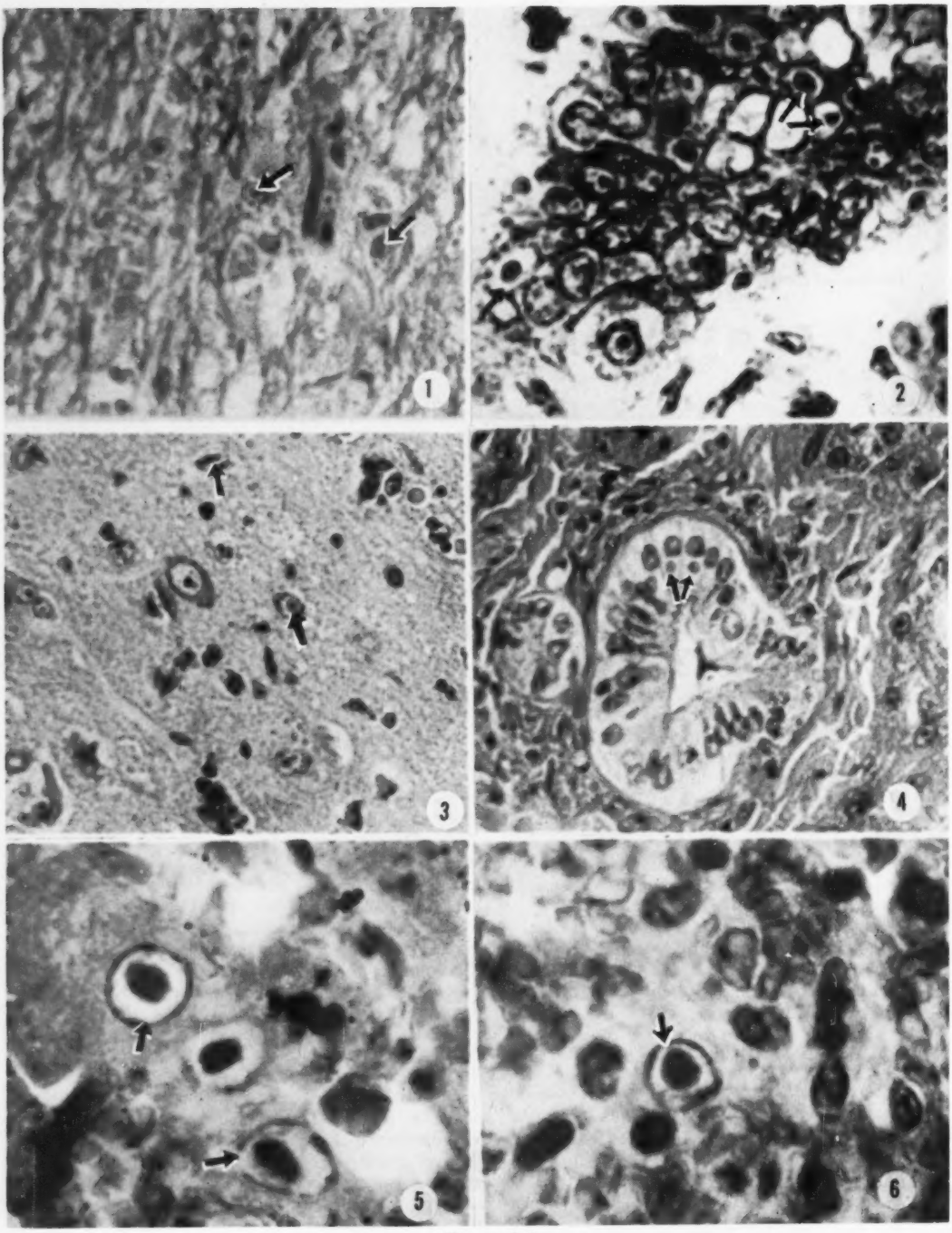
Canine Distemper.—In 1905, it was reported[8] that the causative agent of canine distemper was a filterable virus. Later, other investigators[13,21] confirmed these findings and described the clinical signs and the pathologic changes in this disease.

In this case, submeningeal lesions were found in the cerebellum, with loss of substance, hyperemia, and perivascular cuffing; glial cells contained large, intranuclear inclusion bodies (fig. 1). The stomach of a dog (fig. 2) showed hyperemia and, in the mucosal and gastric gland epithelium, numerous cytoplasmic inclusions were seen.

Raccoon Distemper.—An epizootic of distemper in raccoons (*Procyon lotor*), was reported in Kansas in 1953.[47] In 1948,[20] it was reported that members of the families Canidae, Mustelidae, and Procyonidae were susceptible to canine distemper. This disease was reported in foxes, skunks, and raccoons from Connecticut in 1955.[26]

Microscopically, lesions appeared in submeningeal and subependymal areas of the cerebrum (fig. 3), cerebellum, and medulla. These lesions consisted of loss of substance, areas of degeneration, and necrosis; eosinophilic intranuclear inclusion bodies were seen in the glial cells. Intracytoplasmic and intranuclear inclusions also appeared in the epithelial cells of the lungs, stomach, colon, salivary glands, adrenal glands, and kidneys. Cytoplasmic inclusions were present in the bile ducts (fig. 4), and epithelium of the urinary bladder.

From the comparative pathology section, Division of Research Services of the National Institutes of Health, Bethesda, Md. (Habermann); Mr. Williams is with the Pied Piper Farms, Inc., Newark, Del.; and Dr. Fite, with the Carville Leprosarium, St. Gabriel, La.



Figures 1-6

Infectious Canine Hepatitis.—In 1925, enzootic fox encephalitis or infectious canine hepatitis was first observed in silver foxes.[21] This disease differed from canine distemper in that it usually followed an acute course and secondary infections occurred infrequently. The hemorrhagic nature of the disease was emphasized.[21] An accompanying edema was present in many of the tissues, with fluid in the body cavities and fibrin deposits on the liver.[58]

Microscopically, he liver often had centrolobular lesions and was characterized by necrosis, infiltration of inflammatory cells, hemorrhages, and intranuclear inclusion of the hepatic cells. The inclusion bodies usually had a basophilic tint (fig. 5). The spleen was enlarged and hemorrhagic and showed proliferation of endothelial cells which often contained intranuclear inclusions (fig. 6). Occasionally, the kidney was congested and atrophic glomeruli contained intranuclear inclusions in the glomerular endothelial cells.

Panleukopenia of Cats.—In 1938, a spontaneous infectious disease of cats called "feline agranulocytosis" was reported.[39] The clinical and pathologic changes of the disease were described later.[24,41a] The disease was characterized by panleukopenia, dehydration, enteric lesions, and the presence of intranuclear inclusions in mononuclear cells of the lymph follicles of the mesenteric lymph nodes and in the glandular epithelium of the ileum and colon.

The colon was degenerated and hemorrhagic, with necrosis of the mucosa and submucosa (fig. 7). Intranuclear inclusions were present in the mucous glandular epithelial cells.

Giant Cell Pneumonia of Cynomolgus Monkeys.—Virus pneumonia of human infants was described in 1945,[54] in which the histologic lesions featured multinucleated giant cells containing intranuclear inclusions. Multinucleated giant cells were also found in the lungs of susceptible cynomolgus monkeys inoculated with measles virus.[9] In 1957, giant cell pneumonia was observed in 2 of 60 Cynomolgus monkeys submitted for necropsy.[23]

Interstitial pneumonia of a Cynomolgus monkey with multinucleated giant cells containing intranuclear inclusion bodies in the alveoli is shown (fig. 8).

Sabin B Virus of Monkeys.—This agent was first isolated from a person with acute ascending myelitis following a monkey bite.[60] Since then, the virus has been isolated from the saliva of apparently normal monkeys. In 1935, the histopathology of the experimental disease, after inoculation by various routes in Rhesus monkeys and rabbits, was described in detail.[61] Intranuclear inclusions were demonstrated in the brain, liver, spleen, adrenal glands, ovaries, mesenteric lymph nodes, and skin. In the latter 2 tissues, multinucleated giant cells with intranuclear inclusion bodies were seen. Figure 9 shows the epidermis of a monkey with degeneration of the surface layers and proliferation of the deep layer of stratified squamous epithelial cells and the formation of vesicles. Intranuclear inclusions were seen in the degenerated stratified squamous epithelial cells.

Experimental Rift Valley Fever in Mice.—In 1930, Rift Valley fever, or enzootic hepatitis, was first described as a disease of sheep in the Rift Valley of East Africa.[12] In 1932, sheep, goats, and cattle were found to be readily infected and the most marked gross changes were found in the liver.[18]

Microscopically, there was hyaline degeneration of the liver, extensive destruction of hepatic cells, and infiltration of polymorpho-

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Legends for Figures 1-6 on Opposite Page

Fig. 1—Intranuclear inclusion bodies in canine distemper are shown (arrows) in the glial cells of the white medullary matter of the cerebellum. H. & E. stain; x 1,400.

Fig. 2—Intranuclear and cytoplasmic inclusion bodies in canine distemper are shown in the mucosal and gastric epithelial cells of the stomach. H. & E. stain; x 1,400.

Fig. 3—Intranuclear inclusion bodies in raccoon distemper in the glial cells of the hippocampus. H. & E. stain; x 1,040.

Fig. 4—Raccoon distemper cytoplasmic inclusion bodies are shown in the bile duct epithelial cells. H. & E. stain; x 850.

Fig. 5—Infectious canine hepatitis intranuclear inclusion bodies are shown in the hepatic cells of the liver. H. & E. stain; x 2,000.

Fig. 6—Infectious canine hepatitis intranuclear inclusion bodies are shown in the endothelial cells of the spleen. H. & E. stain; x 2,000.

TABLE 1—Important Viral Diseases in Man and Other Animals; the Animals Are Listed in Order of Susceptibility to Each Disease. The Occurrence of Inclusion Bodies in Animals, Organs, and Tissues Affected Are Also Listed

Disease	Animals susceptible	Intra-nuclear inclusions	Cytoplasmic inclusions	Organs affected	Characteristics
Rabies	Man, dogs, lab. animals, carnivores, herbivores, bats.	−	+ (Negri)	Brain, spinal cord, salivary glands.	Neurotropic.
Pseudorabies	Cattle,* guinea pigs,* mice, man, monkeys,* rabbits,* pigs.*	+	−	Brain, spinal cord, spinal ganglia, heart.	Neurotropic, mesotheliotropic.
Variola (smallpox)	Man, monkeys, sheep, rabbits.	−	+ (Guarnieri)	Skin, lungs.	Dermotropic, epitheliotropic.
Cowpox	Cattle, man, sheep, guinea pigs, rabbits.	−	+	Skin.	Dermotropic.
Sheeppox	Sheep, man, horses, goats.	−	+	Skin.	Dermotropic.
Fowlpox	Birds, mice.	−	+ (Bollinger)	Skin, mouth, nasal sinuses, crop, esophagus.	Dermotropic, epitheliotropic.
Ectromelia (mousepox)	Mice.	−	+	Feet, mouth, tail, liver, spleen, pancreas.	Dermotropic, epitheliotropic.
Rubeola (measles)	Man,** monkeys.**	+**	+**	Skin, respiratory tract.	Dermotropic, epitheliotropic.
Varicella (chickenpox)	Man, monkeys, rabbits.	+	−	Skin, testicles, cornea.	Dermotropic, epitheliotropic.
Herpes simplex	Man, monkeys, guinea pigs, rabbits, mice.	+	−	Skin, cornea, mouth, tonsils, brain.	Dermotropic, neurotropic.
Herpes zoster	Man, monkeys, rabbits.	+	−	Skin, brain, cornea, spinal ganglia, spinal cord.	Dermotropic, neurotropic.
Sabine B virus	Monkeys, man, rabbits, hamsters.	+	−	Brain, meninges, skin, spleen, adrenal glands, lymph nodes.	Neurotropic, Epitheliotropic, dermotropic.
Giant cell pneumonia	Man, monkeys.	+	−	Lungs.	Epitheliotropic.
Yellow fever	Man, monkeys,* mice,* marsupials.	+	−	Kidneys, liver.	Epitheliotropic.
Infectious hepatitis	Man.	−	−	Liver.	Epitheliotropic.
Rift Valley fever	Sheep, cattle, man, mice, ferrets, hamsters, rats (European, African rodents).	+	−	Liver, gallbladder, lymph nodes.	Epitheliotropic, endotheliotropic.
Louping ill	Sheep, man, mice, monkeys, pigs.	−	+	Brain, spinal cord.	Neurotropic.
Bluetongue	Sheep, cattle, goats, mice.	−	−	Lips, tongue, gums, nose, muscles, coronary band.	Endotheliotropic, epitheliotropic.
Distemper	Dogs, foxes, ferrets, raccoons, mink, skunks.	+	+	Brain, urinary tract, gallbladder, lungs, spleen, lymph nodes, stomach.	Neurotropic. epitheliotropic.
Canine hepatitis	Dogs, foxes.	+	−	Kidneys, liver, gallbladder, spleen, bone marrow, brain, testicles.	Epitheliotropic, mesotheliotropic, neurotropic.
Panleukopenia	Cats, lions, tigers, raccoons.	+	−	Ilium, colon, spleen, mesentery, lymph nodes, bone marrow.	Epitheliotropic, mesotheliotropic.
Feline pneumonitis	Cats, mice.	−	+	Nasal passages, conjunctiva, lungs.	Pneumotropic.
Infectious myxomatosis	Rabbits.	−	+	Skin, subcutaneous tissue, spleen, lymph nodes.	Epitheliotropic, mesotheliotropic.
Shope fibroma	Rabbits, squirrels.	−	+	Subcutaneous tissue.	Mesotheliotropic.
Virus III	Rabbits.	+	−	Testes, heart.	Endotheliotropic, cardiotropic.
Equine virus abortion (rhinopneumonitis)	Horses, hamsters.	+	−	Lungs, liver, bile duct, lymph nodes.	Epitheliotropic.
Vesicular stomatitis	Horses, cattle, pigs, guinea pigs.	−	−	Tongue, lips, gums.	Epitheliotropic.
Bovine malignant catarrh	Cattle, rabbits.	−	+	Mouth, nose, eyes, trachea, lungs, lymph nodes, intestines, brain.	Pneumotropic, epitheliotropic, neurotropic.

TABLE 1 (Continued)—Important Viral Diseases in Man and Other Animals; the Animals Are Listed in Order of Susceptibility to Each Disease. The Occurrence of Inclusion Bodies in Animals, Organs, and Tissues Affected Are Also Listed

Disease	Animals susceptible	Intranuclear inclusions	Cytoplasmic inclusions	Organs affected	Characteristics
Infectious bovine rhinotracheitis	Cattle.	–	+	Nasal passages, trachea, bronchi.	Pneumotropic.
Sporadic bovine encephalomyelitis	Cattle, guinea pigs.	–	+	Peritoneum, spinal cord, brain.	Mesotheliotropic, neurotropic.
Mucosal disease	Cattle.	–	+	Mouth, esophagus,* gallbladder, kidneys, colon, lymph nodes.	Epitheliotropic, mesotheliotropic.
Foot-and-mouth disease	Cattle, man, sheep, guinea pigs, goats, reindeer, camels, rabbits, mice,† birds, carnivore.	–	–	Skin, lips, tongue, palate, vulva, udder, teats, feet, conjunctiva, stomach.	Dermotropic, epitheliotropic, myotropic.
Rinderpest	Cattle, goats, rabbits.	–	–	Stomach, lymph nodes, mouth, esophagus, intestines, kidneys, urinary bladder.	Epitheliotropic, mesotheliotropic.
Hog cholera	Pigs.	–	–	Blood vessels, skin, colon, lymph nodes, urinary bladder, brain, meninges.	Endotheliotropic, neurotropic.
African swine fever	Pigs.	–	–	Blood vessels, skin, colon, lymph nodes, spleen, meninges, urinary bladder.	Endotheliotropic.
Teschen disease	Pigs.	–	–	Brain, spinal cord.	Neurotropic.
Vesicular exanthema	Pigs, horses.	–	–	Snout, lips, gums, tongue, feet.	Epitheliotropic.
Swine influenza	Pigs, ferrets, mice.	–	–	Trachea, lungs.	Pneumotropic.
Laryngotracheitis	Chickens, pheasants.	+	–	Larynx, trachea.	Epitheliotropic.
Newcastle disease	Chickens, turkeys, man, mice, hamsters.	–	–	Mouth, lungs, intestines, spleen, brain.	Epitheliotropic, neurotropic.
Owl disease	Owls.	+	–	Liver, spleen.	Epitheliotropic.
Wild rat nephritis	Wild rats.	+	–	Kidneys.	Epitheliotropic.
Mouse myositis (Theiler's‡)	Mice.	+	–	Muscle.	Myotropic.
Mouse hepatoma	Mice.	–	+ (Waxy bodies)	Liver.	Epitheliotropic.
Salivary gland disease§	Man, chimpanzees, guinea pigs, rats, mice, dogs, hamsters.	+	–	Brain, lungs, liver, salivary glands, kidneys, adrenals, heart.	Epitheliotropic, neurotropic.
Lymphocytic choriomeningitis	Mice, rats, man, monkeys, guinea pigs, dogs.	–	–	Meninges, brain, spinal cord.	Mesotheliotropic.
Encephalomyelitis (Theiler's disease)	Mice.	–	–	Spinal cord, spinal ganglia, medulla.	Neurotropic.
Dengue	Man, mice, monkeys.	–	–	Skin.	Dermatropic, neurotropic.
Poliomyelitis	Man, monkeys,*** mice,*** cotton rats.	–	–	Brain, spinal cord.	Neurotropic.
Western and eastern equine encephalitis	Horses, man, monkeys, guinea pigs, goats, cats, pigs, mice, hamsters, ducks, pheasants, turkeys.	–	–	Brain, spinal cord.	Neurotropic.
St. Louis encephalitis	Man, mice, monkeys.	–	–	Brain, spinal cord.	Neurotropic.
Japanese B encephalitis	Man, mice, guinea pigs.	–	–	Brain.	Neurotropic.
Venezuelan encephalitis	Horses, man, rabbits, guinea pigs, mice.	–	–	Brain, spinal cord, tongue.	Neurotropic, epitheliotropic.

*Inclusions formed in these animals or body areas, but not in others. **Inclusions formed in multinucleated giant cells in lungs. †Unweaned mice susceptible. ‡Theiler's (GD VII) virus. §Virus host specific. ***Lansing strain.

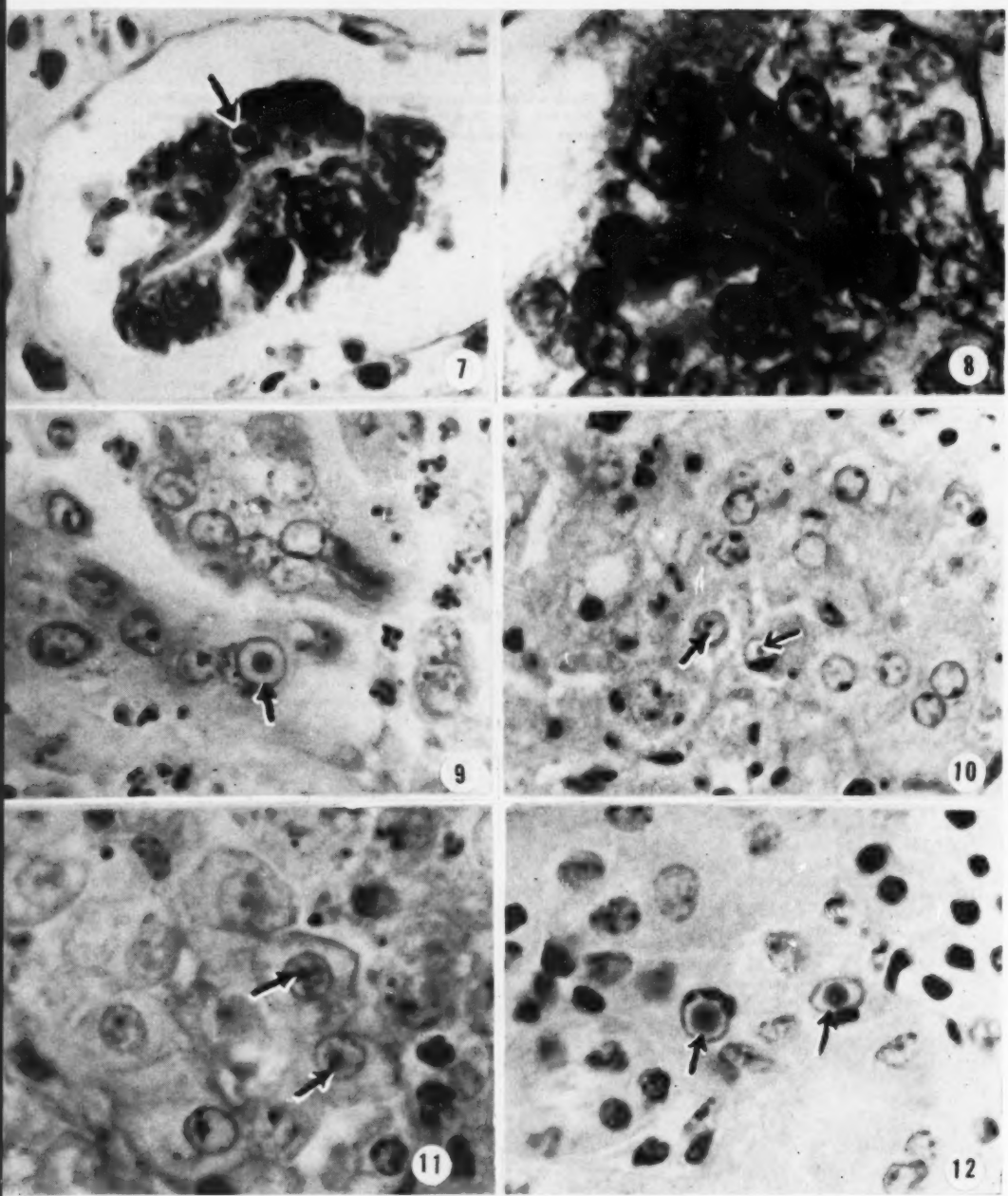

Fig. 7—Intranuclear inclusion bodies are shown in the mucous gland epithelial cells of the colon of a cat with panleukopenia. H. & E. stain; x 1,400.

Fig. 8—Intranuclear inclusion bodies in a multinucleated giant cell of the lung of a monkey with giant cell pneumonia. H. & E. stain; x 1,000.

Fig. 9—Intranuclear inclusion bodies in the squamous epithelial cells of the skin of a monkey with Sabin B virus infection. H. & E. stain; x 2,000.

Fig. 10—Intranuclear inclusion bodies in the hepatic cells of the liver in a mouse with experimentally induced Rift Valley fever. Phloxine methylene blue stain; x 2,000.

Fig. 11—Intranuclear inclusion bodies in the hepatic cells of the liver of a monkey with experimentally induced yellow fever. Phloxine methylene blue stain; x 2,000.

Fig. 12—Intranuclear inclusion bodies in the tubular epithelial cells of the kidney of a monkey with experimentally induced yellow fever. Phloxine methylene blue stain; x 2,000.

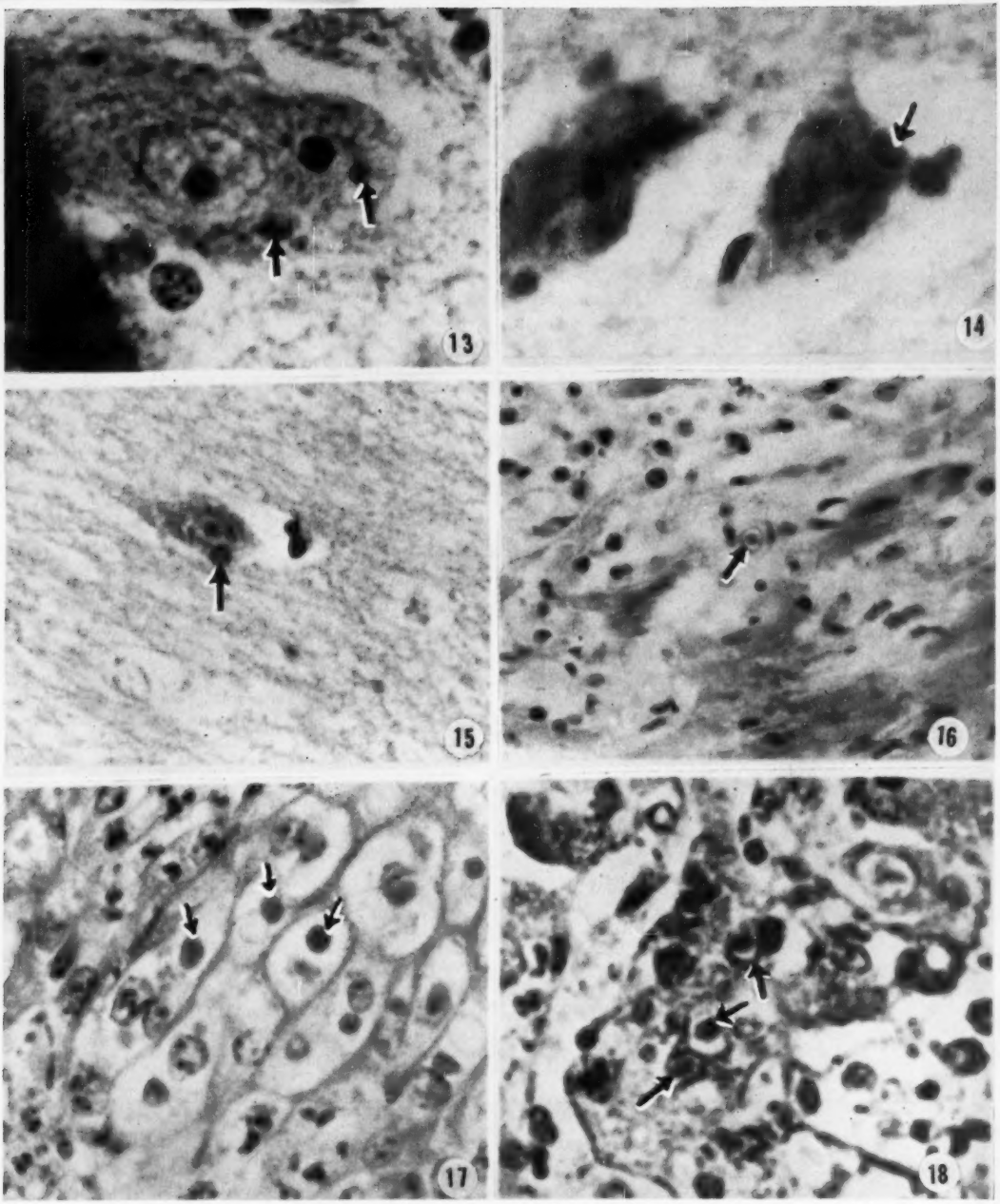

Fig. 13—Human rabies Negri bodies, cytoplasmic inclusion bodies in the Purkinje cells of the cerebellum. Schliefstein; x 2,000.

Fig. 14—Negri bodies in the large pyramidal cells of the hippocampus of a fox with rabies. Schliefstein stain; x 2,000.

Fig. 15—Bovine rabies Negri bodies in the Purkinje cells of the cerebellum. Schliefstein stain; x 1,400.

Fig. 16—Intranuclear inclusion bodies in mononuclear cells of the heart of a rabbit with experimentally induced pseudorabies. H. & E. stain; x 1,000.

Fig. 17—Cytoplasmic inclusion bodies in the squamous epithelial cells of the esophagus of a cow with mucosal disease. H. & E. stain; x 2,000.

Fig. 18—Intranuclear inclusion bodies in the hepatic cells of the liver of a hamster with experimentally induced equine virus abortion. H. & E. stain; x 1,400.

nuclear leukocytes. The chromatin material in some of the degenerated hepatic nuclei appeared clumped and marginated. Small oval homogeneous eosinophilic intranuclear inclusion bodies were seen in the nuclei of these cells (fig. 10).

Experimental Yellow Fever in Monkeys.—In 1928, intranuclear inclusions were described in the liver cells of Brazilian monkeys infected with yellow fever virus.[71] They were comparable to the liver inclusions observed in Rhesus monkeys infected with the West African yellow fever.[65] Other monkeys of Africa and South America were also found to be susceptible to West African yellow fever virus. In 1930, white mice were found susceptible to the virus when injected intracerebrally.[70]

In experimental yellow fever in Rhesus monkeys, there was midzonal hepatic necrosis with hyaline degeneration, but without any alteration of architecture. The hepatic cells of the liver contained intranuclear inclusions which were acidophilic, variable in size, granular in appearance, and irregular in outline (fig. 11). These bodies have been seen only occasionally in human beings with yellow fever. The kidneys of experimentally infected monkeys had nuclear inclusion bodies in the tubular epithelial cells (fig. 12).

Rabies in Man.—The infective agent of rabies was recovered from the brain of diseased animals and from inoculated animals by the intracerebral route.[52] In 1903, characteristic intracytoplasmic inclusion bodies (Negri bodies) in stained microscopic sections of the nerve cells of the brains of man and other animals were described.[49] Negri bodies were found in the cytoplasm of neurons of the hippocampus, the large pyramidal cells of the cerebrum, the Purkinje cells of the cerebellum, and in the large neurons of the basal and cranial nuclei. The cerebellum had areas of hemorrhages and necrosis, and Negri bodies in the Purkinje cells (fig. 13).

Fox Rabies.—An epizootic of rabies in wild foxes was described in which in one area 26 per cent of the brains from 35 foxes contained Negri bodies while, in another area, brains of all of the 59 foxes examined contained Negri bodies.[34,35] There was degeneration, necrosis, and neuronophagia of large neurons of the hippocampus and Negri bodies were seen in the large pyramidal cells (fig. 14).

Bovine Rabies.—The transmission of vampire bat rabies to cattle was observed in the state of Santa Catharina in southern Brazil in 1921.[25] The investigators reported that 2 weeks after bats fed upon the cattle, the cattle would develop a paralysis and die. Rabies virus was isolated from the brains of rabbits and guinea pigs inoculated with brain suspensions from infected cattle. Figure 15 shows a section of the cerebellum from a cow from Trinidad that was bitten by a vampire bat. Areas of degeneration, hemorrhages, and necrosis were seen in the granular and ganglionic layers of the cerebellum, and Negri bodies were seen in the cytoplasm of the Purkinje cells.

Pseudorabies (Mad Itch) in Rabbits.—In 1930, pseudorabies was observed in a herd of 12 dairy cows in Iowa, in which 9 of 12 cows succumbed to the disease.[63] The lesions consisted of degeneration and necrosis of the cortical nerve cells. In 1933, the disease was described in experimentally inoculated rabbits, guinea pigs, monkeys, pigs, and cattle.[28a] It was reported that although lesions were observed in cortical

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Fig. 19—Cytomegalic intranuclear inclusion bodies in the ductal epithelial cells of the submaxillary gland of a guinea pig with salivary gland disease. H. & E. stain; x 1,000.

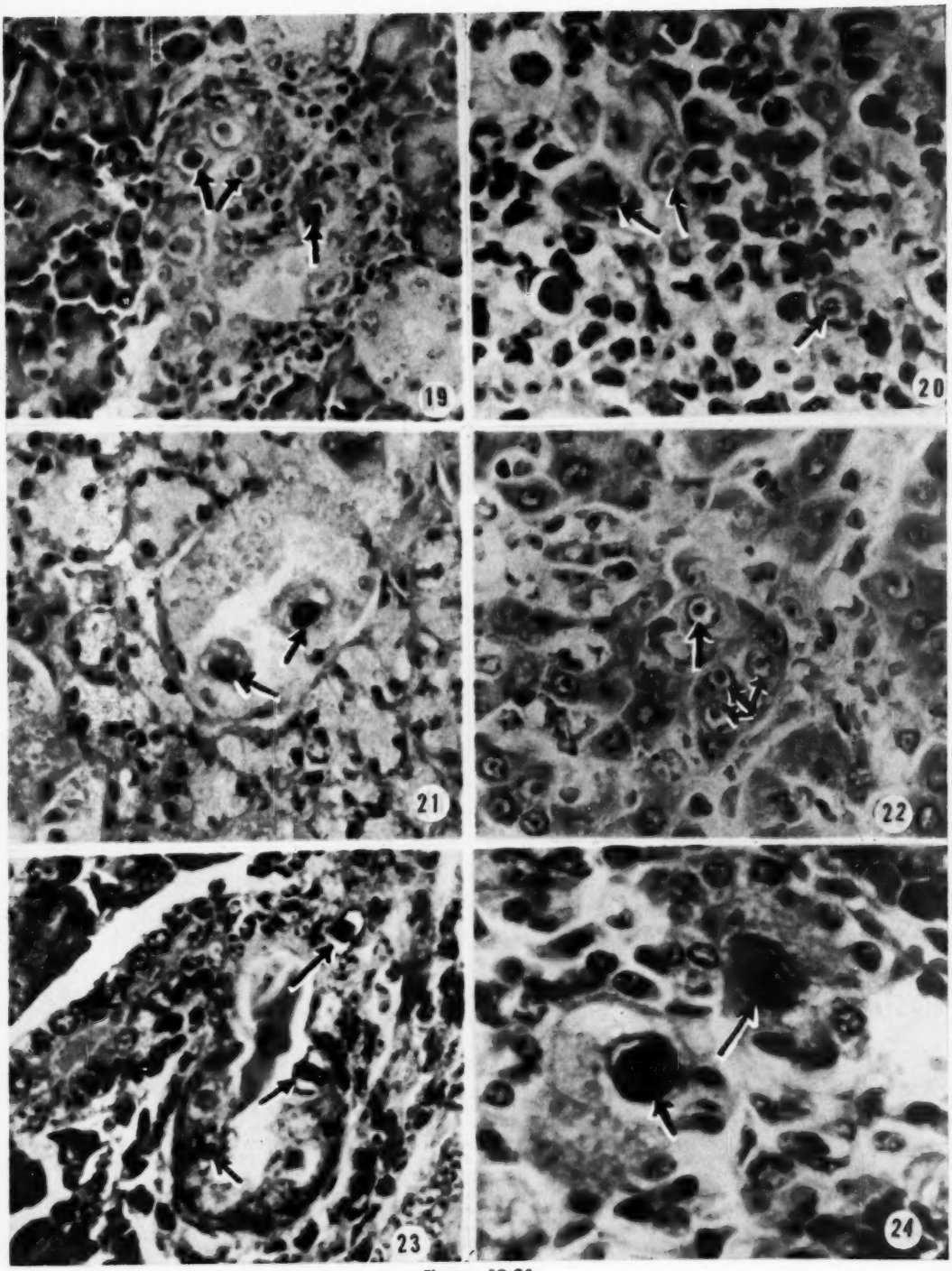
Fig. 20—Intranuclear inclusion bodies in the large mononuclear cells in the meningeal exudate of a guinea pig with experimentally induced salivary gland disease. H. & E. stain; x 1,000.

Fig. 21—Cytomegalic intranuclear inclusion bodies in the ductal epithelial cells of the submaxillary gland of a rat with salivary gland disease. H. & E. stain; x 1,000.

Fig. 22—Intranuclear inclusion bodies in the hepatic cells of the liver of a rat with experimentally induced salivary gland disease. H. & E. stain; x 1,000.

Fig. 23—Cytomegalic intranuclear inclusion bodies in the ductal epithelial cells of the submaxillary gland of a dog with salivary gland disease. H. & E. stain; x 890.

Fig. 24—Cytomegalic intranuclear inclusion bodies in the ductal epithelial cells of the submaxillary gland of a chimpanzee with salivary gland disease. H. & E. stain; x 1,050.



Figures 19-24

nerve cells of all species, nuclear inclusions were seen only in rabbits and guinea pigs. A later report stated that with the phloxine methylene blue stain, intranuclear inclusions in rabbit tissues were seen in all the embryonic layers, as well as, nerve cells, glia, capillary endothelium, sarcolemmal cells, and Schwann cells, and that the inclusions consisted of coarse, pale pink, and occasionally reddish pink granules.[28b]

Other authors also reported that intranuclear bodies were seen in the neurons and glial cells in monkeys, that a few inclusions were present in the cortical cells in cattle, and that intranuclear inclusions were seldom seen in swine. Rabbits inoculated subcutaneously with pseudorabies virus also became infected.[53] By this route, patchy and confluent areas of leukocytic infiltration, proliferation of fibroblasts and mononuclear cells, and necrosis of myocardium were observed. Intranuclear inclusion bodies were seen in the fibroblasts and large mononuclear cells (fig. 16).

Mucosal Disease of Cattle.—The first reported occurrence of mucosal disease was in 1946.[51] Since then, others have reported a mucosal disease complex from 20 states and Canada. Recently, an epizootic in a Hereford-Holstein-Friesian herd in Pennsylvania was described in which inclusion bodies were observed.[68] The pathologic lesions were confined to the epithelium of maxillary sinuses, oral mucosa, esophagus, abomasum, duodenum, cecum, gallbladder, and kidneys. Microscopically, the esophagus showed scattered ulcerated areas, degeneration of the squamous epithelium, and cytoplasmic inclusions in the mucosal epithelial cells (fig. 17).

Experimental Equine Virus Abortion in Hamsters.—In 1940, abortion was produced in mares with filtered material from an aborted fetus.[15] In the same year, the virus of equine influenza was isolated.[66] At that time, intranuclear inclusions were described in the hepatic cells, bile ducts, and the bronchiolar epithelium of an aborted equine fetus.[15] In 1953, the similarity between equine abortus virus and equine influenza virus was demonstrated and it was suggested that the term viral rhinopneumonitis be used for these 2 diseases.[16]

It was observed that newborn hamsters infected with filtrates from aborted equine fetal livers developed areas of focal necrosis and degeneration of hepatic cells.[2] Intranuclear inclusions were observed in these cells (fig. 18).

Salivary Gland Disease in Guinea Pigs.—This disease was first observed in 1920 and the causative agent was described as an intracellular protozoan-like parasite in the ductal epithelium of the submaxillary glands of guinea pigs.[32] In 1926, this disease was identified as a host-specific virus disease of guinea pigs and was transmitted to young guinea pigs by intracerebral inoculation of salivary gland emulsions.[10] In 1939, the pathogenicity of this virus in guinea pigs was described.[57]

Microscopically, the submaxillary glands of infected animals had degeneration of the ductal epithelium, and cytomegalic intranuclear inclusions were present in the ductal epithelial cells (fig. 19). The brains of intracerebrally inoculated animals had hemorrhages of the meninges, infiltration of inflammatory cells, and proliferation of large mononuclear cells. Intranuclear inclusion bodies were seen in large mononuclear cells of the meningeal exudate (fig. 20).

Salivary Gland Disease in Rats.—The first report of the salivary gland virus in rats was from China in 1934.[38] These in-

Legends for Figures 25-30 on Opposite Page

Fig. 25—Cytoplasmic inclusion bodies in the squamous epithelial cells of the foot pad of a guinea pig experimentally infected with cowpox. H. & E. stain; x 850.

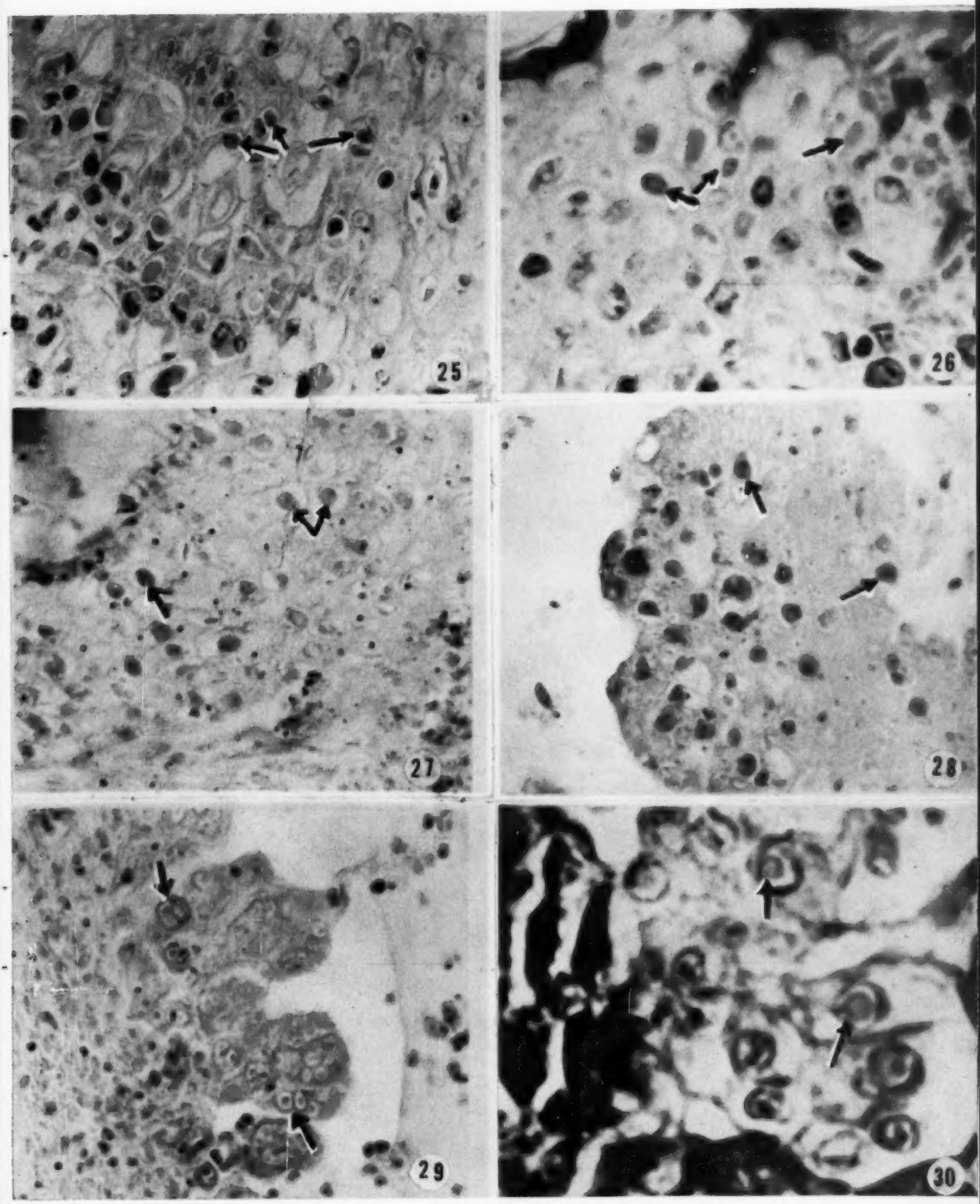
Fig. 26—Cytoplasmic inclusion bodies in the squamous epithelial cells of the skin of a mouse with infectious ectromelia (mousepox). H. & E. stain; x 1,200.

Fig. 27—Cytoplasmic inclusion bodies of fowlpox are shown in the follicular epithelial cells of the skin. H. & E. stain; x 950.

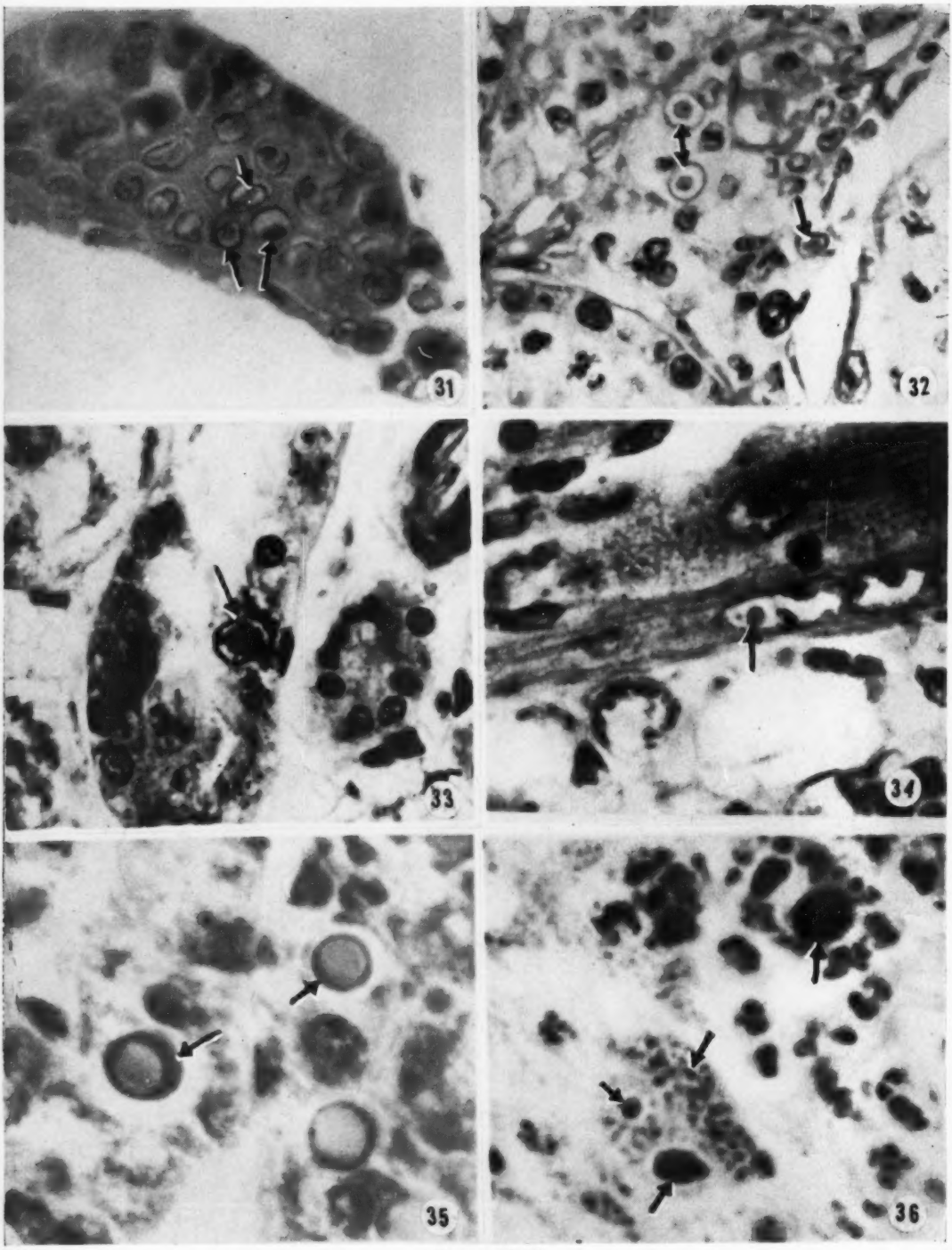
Fig. 28—Experimentally induced fowlpox—cytoplasmic inclusion bodies are shown in the squamous epithelial cells of the nasal mucosa. H. & E. stain; x 850.

Fig. 29—Intranuclear inculsion bodies are shown in the superficial epithelial cells of the trachea of a chicken with laryngotracheitis virus infection. H. & E. stain; x 950.

Fig. 30—Intranuclear inclusion bodies are shown in the epithelial cells of the islets of Langerhans of the pancreas of a chicken with pancreatitis. H. & E. stain; x 1,725.



Figures 25-30



Figures 31-36

vestigators also observed intranuclear eosinophilic inclusion bodies in the submaxillary glands of wild rats, hamsters, and white mice. One year later, rats were experimentally infected with the specific virus for this species and inclusion bodies were seen in the visceral organs.[37] Cytomegalic intranuclear inclusions were seen in enlarged, ductal epithelial cells of the submaxillary gland of a wild rat (fig. 21). In albino rats inoculated intraperitoneally with wild rat submaxillary gland suspension, livers developed scattered areas of hemorrhages and necrosis and, in these areas, intranuclear inclusions were seen in hepatic cells (fig. 22).

Salivary Gland Disease in a Dog.—The first recorded report of a salivary gland disease in the dog was in a 4-week-old male Great Pyrenees pup that was submitted for necropsy in February, 1956. Numerous blood sucking lice, *Linognathus piliferus*, were observed around the head, neck, and abdominal region. The visceral organs were pale, but had no significant lesions. There was degeneration of the ductal epithelium of the submaxillary gland and eosinophilic cytomegalic intranuclear inclusion bodies were seen in these cells (fig. 23).

Salivary Gland Disease in Chimpanzees.—In 1935, intranuclear inclusions observed in a chimpanzee resembled those caused by salivary gland viruses in the parotid and salivary glands of 2 *Cebus fatuellus* monkeys that had received irradiated ergosterol.[11] In 1955, a salivary gland virus disease was observed in 8 of 12 chimpanzees.[73] In these animals, intranuclear inclusions were observed in salivary glands, adrenal cortices, and heart. The submaxillary glands had degeneration of the ductal epithelium and occurrence of basophilic cytomegalic intranuclear inclusions in the enlarged epithelial cells (fig. 24).

Experimental Cowpox Virus in Guinea Pigs.—In 1798, vesicular eruptions on the udders of cows were observed.[33] It is now generally believed that the cowpox of cattle is a modified form of human smallpox, since typical circumcribed cutaneous lesions of cowpox have been produced in heifers and rabbits by inoculation with human smallpox virus. The lesions of cowpox virus were demonstrated by foot pad inoculation of guinea pigs.[17] In guinea pigs, the epidermis had large areas of edema, hemorrhages into the malpighian layer, and numerous intracytoplasmic inclusions in many of the swollen squamous epithelial cells (fig. 25).

Infectious Ectromelia (Mousepox).—Infectious ectromelia of mice was first described in 1930.[44] Since then, the virus has been isolated frequently from laboratory mice in England. In 1953, an epizootic was described in a mouse colony in the United States.[72] In the acute disease, no conspicuous lesions are seen in the animals. The chronic disease is characterized by edema, necrosis, gangrene, and a sloughing of skin of the hindlegs. Microscopically, eosinophilic cytoplasmic inclusions are seen in the necrotic and degenerating squamous epithelial cells of the skin (fig. 26). Occasionally, cytoplasmic inclusions are seen in the fibroblasts and endothelial cells of the subcutaneous tissue, in the surface intestinal epithelial cells, and in the acinar cells of the pancreas of infected mice.

Fowlpox Virus.—Fowlpox virus was first demonstrated in 1902.[45] In 1904, minute, coccus-like structures were seen in smear preparations from the cutaneous lesions of fowlpox.[4] These observations were confirmed and evidence was presented that these minute, spherical, granular Borrel bodies were derived from and formed by the cytoplasmic inclusion bodies of fowl-

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Fig. 31—Intranuclear inclusion bodies are shown in the epithelial cells of the cornea of a rabbit with experimentally induced herpes simplex virus infection. H. & E. stain; x 1,600.

Fig. 32—Intranuclear inclusion bodies in the endothelial cells of the testes of a rabbit with virus III infection. H. & E. stain; x 2,000.

Fig. 33—Intranuclear inclusion bodies in the epithelial cells of the collecting tubules of the kidney of a rat with wild rat nephritis. H. & E. stain; x 1,050.

Fig. 34—Intranuclear inclusion bodies in the myocytes of skeletal muscle of a mouse with Myositis. H. & E. stain; x 2,000.

Fig. 35—Hyaline cytoplasmic inclusion bodies (waxy bodies) in hepatic cells of liver of a hepatitis-infected mouse. H. & E. stain; x 1,725.

Fig. 36—Cytoplasmic inclusion bodies in the fibroblasts of subcutaneous muscle of a rabbit with Shope fibroma. H. & E. stain; x 2,000.

pox.[5] In 1941, studies of the pathologic changes were conducted by inoculations of the virus into the feather follicles of the leg and by intranasal instillation.[50] Microscopically, the skin showed hyperplasia of the follicular epithelium, necrosis, and presence of numerous cytoplasmic inclusion bodies in the follicular epithelial cells (fig. 27, 28).

Laryngotracheitis Virus in Chickens.—This respiratory disease of chickens was first reported in 1925 in a flock of chickens in Rhode Island.[46] Chickens are the usual host, but pheasants are also susceptible to the virus by inoculation or natural exposure.[29] The pathologic changes of the disease have been studied in detail.[62] In our studies, there was edema and cellular infiltration of the mucosa and submucosa of the trachea from 24 to 72 hours after inoculation, and intranuclear inclusions were seen in groups of superficial tracheal epithelial cells (fig. 29). In cloacally vaccinated birds, there was necrosis and desquamation of the mucosal epithelium, and intranuclear inclusions were seen in the degenerated columnar epithelial cells.

Avian Pancreatitis.—In 1947, while conducting investigations on lymphomatosis in chickens, intranuclear inclusions in the epithelial cells of the islets of Langerhans of the pancreas of chickens were observed.[41] In 1951, intranuclear inclusions were seen in the acinar cells of the pancreas of turkeys.[43] The islets of Langerhans of a chicken with intranuclear inclusions in the epithelial cells are shown (fig. 30).

Experimental Herpes Simplex Virus in Rabbits.—In 1921, intranuclear inclusions of *Herpes simplex* virus were first described.[40] It was found that the virus could be transmitted to rabbits by rubbing infected material into a scarified area of the cornea. Microscopically, the corneal epithelium was thickened and intranuclear inclusions were seen in the proliferated epithelial cells (fig. 31).

Experimental Virus III in Rabbits.—In 1923, a new virus disease of rabbits was discovered during an attempt at infecting rabbits with the virus of varicella.[56] One year later, the same agent was encountered while inoculating rheumatic fever material into rabbits.[48] Also, Virus III was found when whole blood from apparently normal stock rabbits was inoculated intratesticularly into rabbits in series. There was a proliferation of endothelial cells in the intertubular spaces of the testes accompanied by edema and hyperemia. Intranuclear inclusions were seen in some of the endothelial cells (fig. 32).

Wild Rat Nephritis.—Intranuclear inclusion bodies were reported in the renal cells of wild rats.[27] In further studies, intranuclear inclusions were seen frequently in the kidneys of London sewer rats.[28] In St. Louis, similar inclusions were observed in the kidneys of wild rats, but attempts to produce the disease in laboratory rats failed when filtrates of infected wild rat kidneys were used.[55] In 1947, an investigation was conducted on 139 wild rats procured from Rochester, Detroit, Denver, and San Francisco.[69] Intranuclear inclusion bodies were found in the renal epithelium of 49 per cent of these animals. The occurrence of inclusions was highest in the kidneys from old urban rats and no inclusion bodies were seen in the kidneys of rats from rural areas, albino rats, or immature rats.

Necrosis, degeneration of the collecting tubules, and intranuclear inclusion bodies were seen in some of the degenerated tubular epithelial cells (fig. 33).

Mouse Myositis.—In 1949, the intramuscular response of mice injected with poliomyelitis, lymphocytic choriomeningitis, eastern equine encephalomyelitis, and 4 strains of Theiler's virus was investigated.[59] One strain of Theiler's virus (OD VII) caused necrosis, regeneration of muscle cells, and infiltration of lymphocytes. In some of the regenerating myocytes of the muscles of the thigh, intranuclear inclusions were seen (fig. 34).

Mouse Hepatoma.—In 1940, cytoplasmic inclusion bodies were reported in hepatomas of the mouse and in nontumorous mouse livers.[6] Attempts to transfer liver tissue containing these bodies to laboratory mice were unsuccessful.[7] It was noticed that these structures were distinct from the inclusion bodies of infectious ectromelia. In a study on the transplantation of spontaneous and induced hepatomas of inbred mice, cytoplasmic hyaline bodies (waxy bodies) in the tumor cells of the liver were frequently observed.[3] Vacuolization of the cytoplasm, and hyaline cytoplasmic inclusions were seen in the hepatic cells (fig. 35).

Shope Fibroma in Rabbits.—In 1932, a transmissible fibroma in the skin of rabbits was reported.[64] The investigator mentioned

the presence of intracytoplasmic inclusion bodies and noted their similarity to molluscum contagiosum. In 1936, red-staining bodies within the cytoplasm of the tumor cells were seen and it was suggested that they represented aggregates of viruses.[31] These inclusions were considered to be protein in nature and degenerative products of the cell cytoplasm or phagocytized material from the surrounding medium.[1] Recently, the nature and staining reaction of the fibroma inclusions were reported.[19] The inflammatory reactions in the subcutaneous muscle and the cytoplasmic inclusions in the proliferating fibroblasts were seen (fig. 36).

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Diagnosis and Surgical Correction of Peritoneopericardial

Diaphragmatic Hernia

in a Dog

D. K. DETWEILER, V.M.D., M.S.
R. S. BRODEY, D.V.M., M.SC. (Vet. Surg.)
G. L. FLICKINGER, V.M.D.

ALTHOUGH the first description of diaphragmatic pericardial hernia in the dog was made in 1811,[7] it was not until 1951, 140 years later, that the first clinical diagnosis of this condition was reported.[11] For the most part, only cases discovered postmortem have been published, and no record of successful surgical correction was found in the literature examined by the authors.

Literature Review

True diaphragmatic hernias are distinguished from false ones on the basis of whether or not a hernial sac is present. The following classification for the various forms has been suggested.[6]

1) *Hernia diaphragmatica vera.*—The abdominal organs pass through an opening in the diaphragm and lie in the thoracic cavity covered by a single or double layer of serosa (hernial sac).

a) Hernia diaphragmatica vera peritonealis (pleuralis or pleuroperitonealis).—The diaphragmatic peritoneum, the diaphragmatic pleura, or both are not ruptured and form a hernial sac for the abdominal organs which have passed into the thoracic cavity.

b) Hernia diaphragmatica paraoesophagealis (intramediastinalis).—The abdominal contents usually pass through an abnormally widened esophageal hiatus and reach a position between the lungs. The mediastinum forms the hernial sac.

2) *Hernia diaphragmatica spuria.*—In this type of hernia, the abdominal organs prolapse through an acquired or congenital defect in the diaphragm and they are not covered by a hernial sac.

a) Hernia diaphragmatica spuria pleuralis.—This is the most common type of diaphragmatic hernia. The displaced organs lie in the pleural cavity.

b) Hernia diaphragmatica spuria pericardialis.—The prolapsed abdominal organs pass into the pericardial sac through a defect in the ventral part of the diaphragm.

A classification, based on pathogenetic principles, for congenital diaphragmatic hernias has been proposed.[3]

1) Deficient closure of the ventral part of the diaphragm.

a) In association with incomplete closure of the pericardial sac.

b) Without other malformations.

2) Abnormally wide hiatus oesophageus.

a) Communication between the abdominal cavity and the pleural cavity.

b) Displacement of the abdominal organs into the cavum mediastini serosum (Sussdorf's space).

The cavum mediastini serosum is a cavity lined by peritoneum which is separated from the omental bursa by the formation of the diaphragm. It is situated between the right mediastinal pleura and the connective tissue layer of the mediastinum, ventral to the aorta and to the right of the esophagus. The latter invaginates the left wall of the space. In the dog, Sussdorf's space extends from the root of the lung to the diaphragm and ends in the region of the esophageal hiatus next to the medial crus of the diaphragm. Normally this space is closed at the diaphragm by a membrane. This membrane can be absent as a result of a congenital defect or can be torn through by sudden elevation of intra-abdominal pressure. In hernias occurring in the region of the esophageal hiatus, the abdominal contents usually pass into Sussdorf's space.

At least 16 cases of diaphragmatic pericardial hernia in the dog have been reported in the literature.[1,2,4,5,7,8,10-12,14] The first description of a single case appeared in 1811[7] and the earliest report found in the American literature was published in 1950.[4] Much of the literature dealing with pericardioperitoneal communications in mammals has been reviewed previously.[9,13]

Of the 16 cases reported, only 3 were diagnosed antemortem.[11,12] One of these dogs showed signs suggesting disease of the gastrointestinal tract (refusal of food, flatulence, sudden crying out in pain, restlessness). In

From the School of Veterinary Medicine, University of Pennsylvania, Philadelphia.

these 3 cases, the abnormality was discovered accidentally. The location of the opening in the diaphragm was in the ventral part (pars sternalis) just over the sternum in 7 of the 9 dogs in which this information was given. In 2 dogs, the prolapse occurred through a widened vena caval hiatus. The contents of the pericardial sac are mentioned for 12 of the cases—in 3, only omentum was found; in 3, only parts of the liver; in 3, only parts of the small intestine; in 2, parts of the small intestine and liver and the entire gallbladder; and in 1, parts of the omentum, liver, stomach, and the spleen. The ages, given for 7 of the animals were: 4 months, 8 months, 1 year (2 cases), 22 months, 6 years, and 8 years. In the 8-month-old dog, spontaneous reposition of the abdominal contents occurred. Surgical correction was attempted in 1 of the dogs at 1 year of age and failed because of adhesions attaching the intestine to the pericardium. Six years later, this dog was still in good health.[12] The third dog in which this condition was diagnosed antemortem was destroyed because of a rupture of an intervertebral disk and resultant paralysis. The sex of 7 of the 8 dogs for which it is given was male.

Case Report

History.—A male, English Setter pup developed normally until it was 5 weeks old, when it began vomiting after meals. Two days after the onset of vomiting, watery diarrhea was noted.

Clinical Examination.—At the time of the first clinical examination (Nov. 30, 1957) the pup was active, eating well, and exhibited no evidence of cardiac or respiratory difficulty. On auscultation of the thorax, the heart sounds were muffled and distinct peristaltic sounds could be heard. The latter were of about the same intensity as when these sounds are heard over the abdominal cavity (fig. 1A). An electrocardiogram taken at the time showed deflections of abnormally low amplitude, especially in the precordial leads (fig. 1B). Lateral and dorsoventral thoracic radiographs (fig. 2A, 3A) revealed a markedly enlarged cardiac silhouette. In the lateral view, loops of bowel could be seen clustered about the heart. Radiographs taken after the oral administration of liquid contrast medium revealed distinctly outlined loops of small intestine within the pericardial sac (fig. 2B, 3B). The radiologist* reported that approximately the distal half of the jejenum and proximal half of the ileum appeared to be herniated into the pericar-

*Dr. Harker Rhodes, assistant professor of radiology, School of Veterinary Medicine, University of Pennsylvania, Philadelphia.

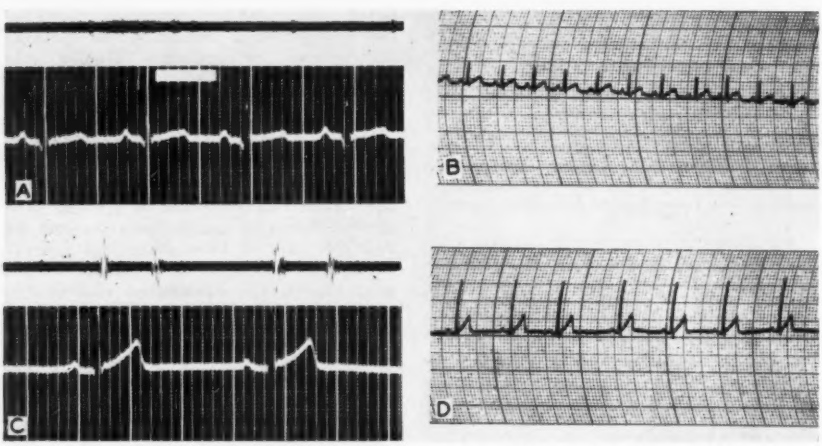

Fig. 1—Phonocardiograms and electrocardiograms taken before (A and B) and after (C and D) surgical correction of the peritoneopericardial diaphragmatic hernia. (A) Simultaneous electrocardiogram (lead 3) and phonocardiogram showing muffled heart sounds and intestinal sounds during first third of record. (B) Lead CL6LL (precordial electrode at sixth left intercostal space at the edge of the sternum paired with an indifferent electrode on the left foreleg) electrocardiogram showing relatively low potentials. (C) Simultaneous electrocardiogram (lead 3) and phonocardiogram. Both records taken at the same amplification as in A. Notice distinct heart sounds and increase in amplitude of electrocardiographic potentials. (D) Lead CL6LL electrocardiogram showing increase in potential following surgery.

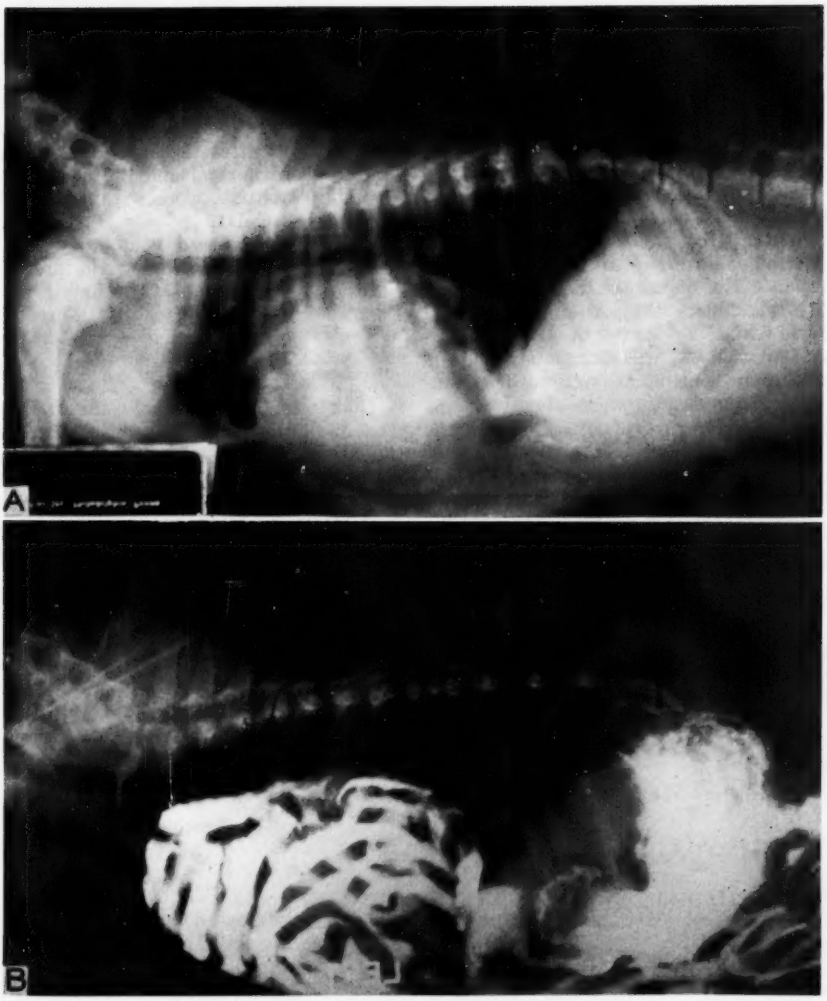

Fig. 2—Lateral thoracic radiograph (A) showing enlarged cardiac silhouette containing radiolucent areas caused by loops of small intestine. Radiograph (B) showing the same condition following administration of barium sulfate solution.

dial space. On the basis of these findings, a diagnosis of hernia diaphragmatica pericardialis was made and surgical correction elected.

Surgical Procedure.—On Dec. 9, 1957, the patient was prepared for surgery and anesthetized with sodium pentobarbital administered intravenously following subcutaneous injection of 0.6 mg. of atropine sulfate. An incision was made through the right eighth intercostal space. Following incision of the parietal pleura, oxygen was administered through an endotracheal tube by manual compression of a rebreathing bag. The pericardial sac was greatly distended and coils of intestine were visible through this transparent serous membrane. The parietal pericardium immediately ventral to the phrenic nerve was incised in a dorsoventral direction. The hernial contents consisted of almost all the mobile portion of small intestine, an enlarged mesenteric

lymph node, and some omentum. The defect in the diaphragm, approximately 2.5 cm. in diameter, was located on the midline, just dorsal to the xiphoid cartilage. The circumference of this defect was smooth, somewhat thickened, and indurated.

A counter incision was made in the muscular portion of the diaphragm and the prolapsed intestines were readily pulled back into their normal position. The diaphragmatic defect was closed with Lembert and simple interrupted stitches using atraumatic No. 00 catgut. Several of these sutures included the adjoining ventral thoracic wall so that the pericardial-peritoneal opening was completely obliterated. Simple continuous sutures of atraumatic No. 00 catgut were placed in the counter incision previously made in the diaphragm, as well as in the dorsal half of the incised pericardial sac. The ventral half of the pericardial incision was left open to prevent accumulation of fluid, and possible cardiac tamponade. The thoracotomy incision was closed in a conventional manner. As soon as the air was aspirated from the pleural cavity, the dog began to breathe spontaneously and an uncomplicated recovery period followed.

Postoperatively, the dog's appetite was good and the vomiting and watery diarrhea, noted preoperatively, were not observed.

Four days after the operation, the heart sounds were normal and the electrocardiographic potentials were within the normal range (fig. 1C, D).

The dog recovered completely and has remained in good health to the present (2 years).

Discussion

Herniation of abdominal contents through the diaphragm into the pericardial cavity is extremely rare, in contrast to herniation into the pleural space. Because of its rarity, clinical diagnosis has been seldom made and an attempt at surgical correction has been reported in the dog only once previously. Similarly, in man this type of hernia is rare and the first successful surgical

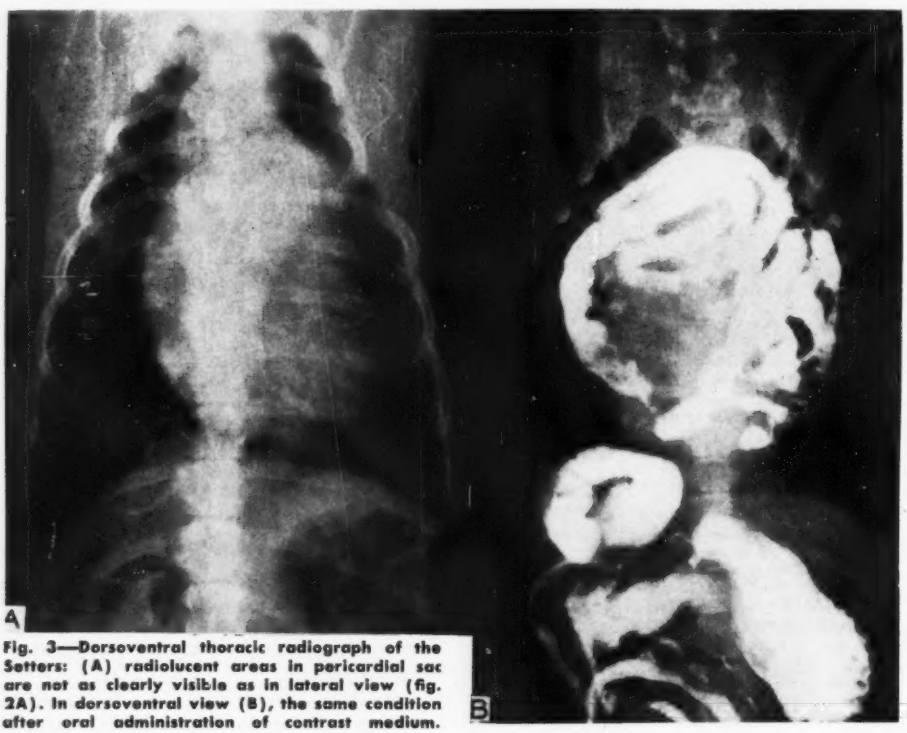

Fig. 3—Dorsoventral thoracic radiograph of the Setters: (A) radiolucent areas in pericardial sac are not as clearly visible as in lateral view (fig. 2A). In dorsoventral view (B), the same condition after oral administration of contrast medium.

correction was reported only recently (1947).[15]

Although explanation of the primary embryologic etiology of these congenital defects must remain speculative, review of the development of the structures involved suggests possible mechanisms to account for the anomaly.[16]

In the developing embryo, the septum transversum initiates the division of the coelom into the thoracic and abdominal cavities and comes to form the ventral portion of the diaphragm. Complete separation of the pericardial, pleural, and peritoneal portions of the coelom is accomplished by growth of the paired pleuroperitoneal and pleuropericardial folds which finally meet and fuse with one another and with the septum transversum. At the time this occurs, the pericardial cavity is still entirely ventral to the pleural cavity and its wall is not separated completely from the body wall. As the lungs grow, the pleuropericardial folds are bent ventrally. Lung growth continues and the separation between the body wall and pleuropericardial folds is extended until these folds fuse ventrally forming the definitive pericardium which isolates the heart from the lungs. The pleuropericardial folds also converge behind the heart, separating the pericardium from the primitive diaphragm. In the dog, the ligamentum diaphragmaticopericardiacum, connecting the pericardium with the diaphragm, is formed where the developing pleuropericardial folds converge and meet in the region of the ventral portion of the diaphragm. The final form of the diaphragm is not achieved until a peritoneal fold grows upward from the ventral body wall and separates the liver entirely from the diaphragm.

It is apparent that part of the substance of the original septum transversum is incorporated in the developing pericardial wall and its thickness may be further reduced as the peritoneal folds grow between it and the liver. If either one or both of these invading folds should deviate sufficiently, the pericardial and peritoneal septa could meet, and local interference with tissue growth would allow communication between the pericardial and peritoneal cavities to develop and persist.

Although herniation of abdominal contents through a peritoneopericardial communication may be fatal, for obvious reasons, several reported cases indicate that little disturbance may occur. Clinically, there may be signs of abdominal discomfort or cardiac embarrassment. In thoracic radiographs, the presence of an enlarged cardiac silhouette in which mottled radiolucent areas are present, delimited by the pericardial sac, should suggest the possibility of diaphragmatic pericardial herniation of intestinal loops. The use of contrast medium can confirm the diagnosis. Muffled heart sounds, low amplitude precordial electrocardiograms, and loud intestinal sounds (borborygmus) over the cardiac region are other signs which may be present. If only the omentum or lobes of the liver enter the pericardial sac, clinical diagnosis is more difficult.

In congenital diaphragmatic hernias, especially those in Sussdorf's space and in the pericardial sac, clinical signs may be absent or misleading and can only be diagnosed through radiographs.[12] Intermittent herniation into the pericardial sac or into Sussdorf's space is possible, occurring transiently and disappearing entirely. In the case reported here, the initial clinical signs suggested an intestinal disturbance or foreign body rather than diaphragmatic hernia.

Summary

A total of 16 cases of peritoneopericardial diaphragmatic hernia in the dog have been reported since the first case was described in 1811. The first clinical diagnosis of this condition was reported in 1951. The clinical diagnosis and surgical correction of an additional case in a 5-week-old pup is described.

It is apparent that this rare condition results from failure of complete separation of the pericardial and peritoneal portions of the embryonic coelom by the pleuropericardial folds.

The clinical signs observed in this type of hernia may suggest an intestinal disturbance. Muffled heart sounds and low amplitude electrocardiographic potentials occur owing to the presence of fluid and abdominal organs in the peritoneal cavity. Thoracic radiographs or surgical intervention are necessary for final diagnosis.

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Reported Incidence of Rabies in the United States—1938-1959

Year	Dogs	Cattle	Horses	Sheep	Swine	Cats	Goats	Misc.	Man	Total
1938	8,452	413	32	164	42	207	11	44	47	9,412
1939	7,386	358	36	17	38	269	10	172	30	8,316
1940	6,194	326	25	53	71	260	4	277	28	7,238
1941	6,648	418	39	68	159	294	9	212	30	7,877
1942	6,332	288	15	48	32	250	12	160	28	7,165
1943	8,515	349	35	45	60	316	19	310	41	9,690
1944	9,067	561	32	40	43	419	14	311	53	10,540
1945	8,505	487	46	11	30	466	10	373	35	9,963
1946	8,384	962	44	15	22	455	12	956	22	10,872
1947	6,949	766	40	15	20	393	9	728	26	8,946
1948	6,610	599	34	14	36	378	5	819	13	8,508
1949	5,237	639	24	22	54	413	6	1,192	10	7,597
1950	4,979	948	33	48	85	428	5	1,375	9	7,910
1951	5,194	821	34	35	53	480	4	1,387	14	8,022
1952	5,261	916	38	19	31	486	7	1,674	21	8,453
1953	5,688	1,012	21	42	38	538	5	1,479	14	8,837
1954	4,083	930	29	23	40	462	10	1,697	8	7,282
1955	2,657	835	33	18	30	343	8	1,915	5	5,844
1956	2,592	700	30	22	30	371	12	2,079	10	5,846
1957	1,908	654	24	13	16	386	12	1,989	6	5,008
1958	1,643	660	31	22	20	353	4	2,075	6	4,814
1959	1,124	700	22	8	18	310	4	1,925	6	4,117
TOTAL:	123,408	14,342	697	762	968	8,277	192	23,149	462	172,257

—Animal Disease Erad. Div., ARS, USDA, March 17, 1960

Rabies Discovered in Greenland

Matthew JENKINS, D.V.M.
Kjeld WAMBERG, D.V.M.

THIS REPORT describes the first confirmed laboratory diagnosis of rabies in animals in Greenland. Rabies in man has not been reported in that country but, due to the close association of dogs and man, it may have occurred.

It has been argued that rabies is unlikely to occur in man in cold climates because the heavy clothing worn most of the time would prohibit saliva from entering wounds inflicted by rabid animals. The validity of this argument has not been determined.

History

During the fall of 1958, we examined 11 dogs, 6 of which had been bitten by other dogs and 5 by foxes. No signs of rabies appeared in any of the 11 dogs during the 14 days they were quarantined.

Greenland is heavily populated with foxes which have moved near military bases in search of food. When the potential health hazard of foxes was realized, the Greenland Health Department made available all of its information on diseases transmissible from animals to man, including incidence of rabies in arctic climates, clinical signs, course, predisposition, prevention, and treatment.

This information included reports on a disease characterized by fits (and called "fits") which had been recognized for the past 100 years. Since 1906, periodic studies had been made of this condition, but no diagnosis of rabies was ever confirmed.

Although goverment officials of Greenland stated that rabies was not known to occur in that country, they recommended treating animal bites as though rabies were involved. All base personnel were warned that foxes might be rabid.

On Nov. 24, 1958, a fox which attacked a team of sled dogs was killed and the brain examined for rabies lesions. No Negri bodies were found. A brain suspension was injected into 3 mice, 1 of which died 12 days following injection; the other 2 died 18 days later. No Negri bodies were found in the 3 mice. Shortly after this negative report was received, the 5 dogs which had been attacked by the fox showed signs of rabies and died within a short time. It was impossible to retrieve the heads of the dogs, because the Greenlanders had burned the bodies.

On Dec. 22, 1958, another dog was bitten by a fox and the fox's head was submitted for laboratory examination. No Negri bodies were found in the brain, nor in the brains of mice which were subsequently inoculated with a suspension of the fox's brain.

In February, 1959, we were informed by the Danish health inspector that fits in foxes and dogs had reached epizootic proportions in some areas and threatened the existence of the native Greenlanders. In northern Greenland, the people are dependent on the services of their sled dogs to secure food, and they are with their dogs most of the time. Although fits had existed in their dogs for many years, the Greenlanders seemed to have adjusted to this problem by immediately killing and burning any dog at the first signs of this condition.

During the latter part of February, 1959, a research project was initiated to ascertain the cause of fits, which clinically resembled rabies.

Methods and Results

Specimens 852, 853, and 854.—On May 10, 1959, 3 heads of animals which had had fits were submitted for laboratory examination. These heads were marked 852-black dog, 853-brown dog, and 854-silver fox. The excised brains were placed in separate Petri dishes and stored at 4 C. The following day, suspensions were made from

Dr. Jenkins, Animal Industry Division, Bureau of Poultry Inspection for California, Monterey Park. Dr. Wamberg is associate professor, Royal Veterinary and Agriculture College, Copenhagen, Denmark, and veterinary adviser to the Greenland Department, Copenhagen, Denmark.

The information in this article was obtained during 1958 and 1959 while Dr. Jenkins was stationed at Thule Air Base, Greenland.

The authors acknowledge the cooperation of the Greenland Government, through the efforts of Dr. I. Giuskud. Further acknowledgement is made of the laboratory assistance provided by the Virus and Rickettsia Laboratory, U. S. Public Health Service, Montgomery, Ala.

The contents of this article reflect the authors' personal views and are not to be construed as a statement of Air Force policy.

portions of each brain in 10 per cent egg yolk and buffered water to which 1,000 units of penicillin and 2 mg. of streptomycin were added. These were centrifuged at 1,000 r.p.m. for 10 minutes. Each supernatant fluid was inoculated intracerebrally into 5 mice. On May 28, 1959, examinations of brain smears of the mice's heads were made by the fluorescent antibody technique. By this method, 852-black dog was negative, 853-brown dog was suggestive of, and 854-silver fox was definitely positive for rabies.

At the end of 21 days, the mice inoculations had the following results: When brain suspension from dog 852 was inoculated into test mice, all mice survived for 21 days. On postinoculation day 11, 1 had a slight hump but later improved in appearance.

When brain suspension from dog 853 was inoculated into test mice, all 5 mice remained normal until postinoculation day 11 when 3 mice became slightly humped. On days 12, 13, and 14, these 3 became definitely humped; 1 of these was paralyzed on day 15, and it was killed. On necropsy, atypical Negri bodies were observed. The second mouse was still humped on day 15 and paralyzed on days 16 and 17, when it was killed. Use of Seller's stain revealed more advanced typical Negri bodies present in large numbers. Test mouse 3 was humped through day 16, paralyzed on day 19, and dead on day 20. Use of Seller's stain again revealed typical Negri bodies. These 3 mice were checked by fluorescent antibody technique with positve results. The remaining 2 mice had only a slight hump from day 12 to day 15, and became normal in appearance after that and remained normal through day 21.

When brain suspension from fox 854 was inoculated into test mice, they were normal until postinoculation day 7, when 2 became irritable. By day 10, all 5 mice were definitely humped. On day 11, 3 were paralyzed; they died on day 12. The remaining 2 were paralyzed and died on day 13. Negri bodies were demonstrated on Seller's stained smears from all these mice.

Specimen 101.—On June 11, 1959, an arctic fox attacked a slowly moving truck in a doglike fashion, biting at the tires and bumper of the vehicle. Two men standing nearby were attacked after attempting to stop the fox's attack on the vehicle. Although the fox was killed during the struggle, 1 of the men was bitten (he was given Pasteur treatment). The fox's brain was examined for Negri bodies which were found on Seller's stained smears. Mice which were subsequently inoculated with a suspension of the positive specimen succumbed in 16 days and were positive for Negri bodies.

Specimen 102.—The dog from which this specimen was obtained had been bitten May 28, 1959, by a teammate which died from a disease resembling rabies. On June 12, 1959, an abrupt change in behavior, characterized by frequent attempts to attack the owner and biting at himself, was observed. The dog was alert, hypersensitive to any moving object, and resented confinement. While being transported to the base by helicopter, he attempted to attack the aircraft. These attacks were followed by periods of staring into space, severe hypersalivation, restlessness, irritability, and protrusion of the membrana nictitans.

Forty-eight hours after the onset of clinical signs, the dog appeared emaciated and dehydrated. He made frequent attempts to eat and drink. He would grasp food, but was unable to swallow it. Also, he bit at himself.

After 72 hours, the dog appeared depressed. His eyes were coated with mucopurulent exudate, saliva continuously drooled, and he was unable to close his mouth. Periodic intervals of completely opening his mouth and scratching at it as though a foreign body were in it were followed by a return of the mouth to its half-closed position. At 96 hours, clinical signs were more pronounced. By hour 120, the membrana nictitans covered 50 per cent of the eyeball and the mouth was open and dehydrated. Frequent attempts to vomit resulted in expulsion of a small amount of clear water-like fluid. The tongue was dry and leathery and areas of necrosis were observed along the borders. The dog was weak and his depression had become much more severe after 120 hours. Emaciation and dehydration were pronounced. Although the dog tried to remain on his feet, by hour 132 he lay for long periods in lateral recumbency. When he sat, his head would gradually fall to the floor or the wall as though he were sleeping. The contact of his head with an object seemed to stimulate his alertness and immediately he would show restlessness. Then, he would return to his recumbent position. At hour 144, the dog was unable to right himself. Although paralysis had progressed to the

point where he could barely lift his head, the dog would still try to bite if stimulated by touch. At hour 146, the dog was barely breathing; 10 minutes later he died. His brain was positive for Negri bodies. Inoculated mice were also positive for Negri bodies.

Following the initial positive laboratory results, the Greenland Government was informed of our findings and a rabies control program was established.

Summary

Rabies, previously unreported in Greenland, was confirmed in 2 dogs and 2 foxes. Human rabies has never been reported in Greenland. Prior to this time the Greenland Health Department recognized a disease among dogs and foxes which they called "fits." Since 1906, periodic studies had been made of this condition, and it was not thought to be rabies.

During the early part of 1959, this disease in dogs reached epizootic proportions in some parts of Greenland and threatened the existence of residents because of their dependence on their dogs for transportation and hunting.

On May 10, 1959, a dog and fox, which showed clinical signs resembling fits and rabies, were found to be positive for rabies by the fluorescent-antibody technique. Negri bodies were found in the brain of the fox, and mice which were inoculated with a brain suspension from the dog and fox were also positive for rabies. Later, another dog and fox were shown to have had rabies. The disease, known since 1906 as fits, proved to be rabies.

Although a rabies control program for Greenland has been instituted, it is unlikely that it will be effective for some time because of the heavy population of foxes.

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Antibiotics for the Control of Vibriosis of Ewes

Highly significant reductions in the rate of abortion among pregnant ewes, orally infected with *Vibrio fetus*, resulted from (1) feeding 80 mg. of chlortetracycline per ewe daily from 27 days prior to inoculation until they lambed; (2) two daily injections of penicillin and dihydrostreptomycin on postinoculation days 5 and 6 and daily feeding of 80 mg. of chlortetracycline per ewe from postinoculation day 5 until lambing; and (3) oral inoculation with *V. fetus*-infected tissues during the preceding pregnancy while the ewes were being continuously fed chlortetracycline.

The results agree with a previous report that feeding chlortetracycline continually at the rate of 80 mg. per ewe daily results in a highly significant reduction in abortions among ewes infected orally with *V. fetus*. They also indicate that such ewes may become immune even though they do not abort. The date indicates that antibiotic therapy may be of value in a flock infected with vibriosis. *—F. W. Frank, W. A. Meinershagen, L. H. Scrivner, and J. W. Bailey: Antibiotics for the Control of Vibriosis of Ewes. Am. J. Vet Res. 20, (Nov. 1959): 973-976.*

Certification of Swine Herds

as Virus Pneumonia-Free

George A. YOUNG, D.V.M.
Norman R. UNDERDAHL, M.S.

VIRUS PIG PNEUMONIA (VPP) has become a major disease of U.S. swine and often makes the difference between profit and loss to swine raisers throughout the nation. Few swine die from the disease, but VPP causes severe economic loss because it increases the total feed required to grow pigs to market weight. The virus persists for long periods within the lungs of infected swine, which results in continuous enzootic infection within affected herds. Antibody development is not stimulated by infection. No drug has been effective for treatment. A general review presented previously outlines the dilemma of VPP and the limited possibilities of its control.[3]

An effective and practical means to eliminate VPP from swine has been recently described.[6] In essence, aseptic pigs are obtained by hysterectomy, raised in isolation, grown to maturity under farm isolation, and used to produce VPP-free pigs by normal birth and production. These second-generation pigs are used to restock other farms. All farms are managed to continuously isolate "disease-free" pigs from those which do not originate in a similar manner. These methods constitute "swine repopulation."

With an effective means of eliminating VPP from swine herds, it becomes possible to establish VPP-free herds which may serve as source stocks to set up additional VPP-free herds. Since such source stocks will undoubtedly sell at a premium, some stock will be promoted as VPP free which is not. The purpose of this communication is to describe studies made to serve as a basis for certification by veterinarians that herds are free of VPP.

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The authors thank the George A. Hormel Co., Fremont, Neb., for their assistance in examining lungs in their plant.

Methods

The lungs of 700 market pigs were examined for evidence of gross lesions at a packing plant.[1] Approximately 100 lungs were examined each month from October, 1958, through April, 1959. Every fifth lung was examined so that the sample each month was from a minimum of 500 market pigs. The 700 samples thus represent a population of at least 3,500 pigs over a 7-month period.

Lungs were graded as VPP-positive, suspicious, or VPP-negative. Typical lesions consisted of plum-colored to reddish brown consolidated areas usually on the lower portion of the apical and cardiac lobes (fig. 1). Lungs that were discolored or abnormal in any way were treated as suspicious. Only those lungs completely normal in gross appearance were listed as negative.

Histologic studies were made from 500 of the lungs sampled. Tissue samples were taken from obvious lesions in VPP-positive or suspicious lungs. When no lesion was present (VPP-negative lungs), a tissue sample was taken from the outer edge of the right apical lobe which is the lobe most apt to show VPP lesions. Tissues were fixed in neutral 7 per cent formalin, embedded in paraffin, and stained with hematoxylin and eosin. The typical histopathologic lesions associated with VPP have been described previously.[2]

Results

The results of the gross and microscopic observations are shown (fig. 2). In approximately 40 per cent of the lungs ex-

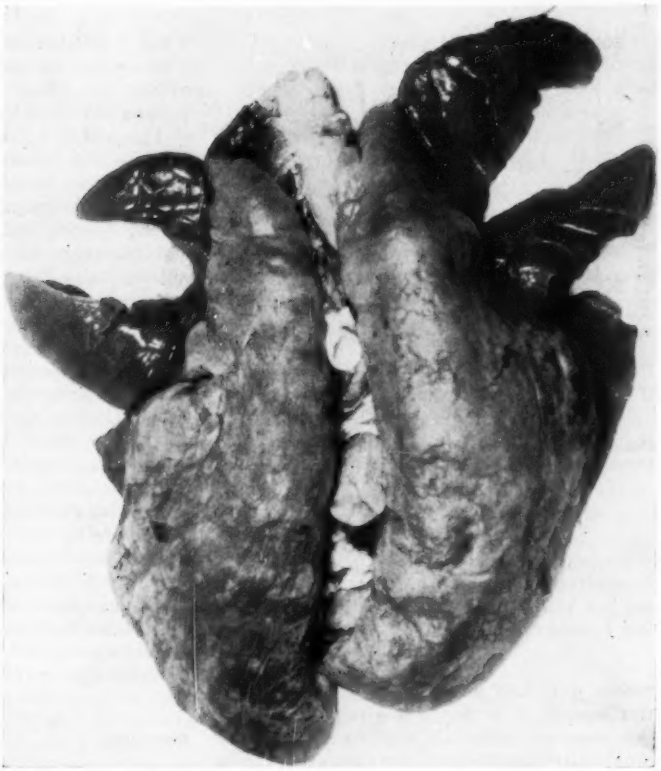

Fig. 1—Lung from a market pig, with plum-colored consolidation typical of virus pig pneumonia.

amined, VPP lesions were present on gross observations. A slightly higher percentage of lungs were VPP-positive on microscopic examination. This was especially true during the early part of the study. Only a small percentage difference was observed in December, January, and February.

Many lungs were examined which were discolored a reddish brown with occasional diffuse pneumonic areas. The appearance of these lungs was distinct from those with VPP but they were classed as suspicious for convenience. Microscopically, this unknown disease was distinct from VPP and presented an interstitial pneumonia rather than a small and large round cell perivascular and peribronchiolar cuffing characteristic of VPP.

The incidence of VPP, based on number of gross VPP-positive examinations, differed slightly from the incidence based on microscopic VPP-positive examinations. Only 3 per cent of the lungs considered positive on gross examination were negative microscopically. About 4 per cent of those lungs designated grossly as VPP-positive were afflicted with another disease.

Errors of greater magnitude were made in cases of grossly classified VPP-negative lungs. This was expected, as early lesions may be detected microscopically before the disease develops sufficiently to cause gross lesions. Lesions caused by diseases or conditions other than VPP were more common in the gross VPP-negative category.

Approximately 22 per cent of the lungs placed by gross examination in a questionable category had lesions typical of VPP. Half of these were microscopically an interstitial pneumonia distinctly different in character from VPP. A lesser percentage disclosed no microscopic abnormalities.

Certification Standards

The practical question which seems answerable from the above data is: How many pigs from a herd should be examined in order to certify a herd as free of VPP?

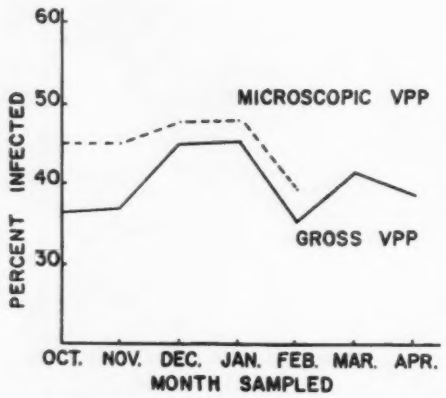

Fig. 2—Incidence of virus pig pneumonia in market pigs at slaughter by gross and microscopic examination.

Working from a desire to include at least 1 positive animal in a herd sample 99 times out of 100 (99% probability), the following formula is useful:

$$n = k\ (r + \tfrac{1}{2})$$

where n is the number to be observed in the sample, $k = \log_e\ (1 + p)$ where p is the desired probability of occurrence of only individuals that are negative in a group of n individuals; $r = a/b$ where a represents the proportion of animals that are negative and b represents the proportion of animals that are positive.

If we assume a herd of 200 animals, with $a = .60$ or the proportion negative and $b = .40$ for the proportion positive* and want a 99 per cent probability, then $k = 4.61$ as determined previously* where k is minus $\log_e\ (1 - p)$ and p is the desired probability level.**

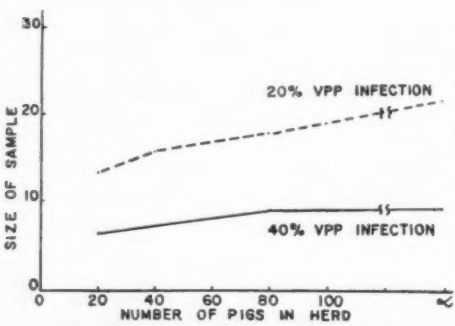

Fig. 3—Theoretic curve showing the sample size required to detect a virus pig pneumonia-infection in a specific pathogen-free swine herd at a 99 per cent probability level.

The above formula has been used to determine the number of pigs which should be examined to assure detection of VPP in a herd. Theoretical values based on herd sizes of 20, 40, 80, and 100 pigs with levels of VPP infection at 20 and 40 per cent are shown (table 3). Assumption of 40 per cent incidence within a VPP-infected herd is conservative.[1] Examination of 7 pigs out of a herd of 20, 8 out of 40, and 9 from herds of infinitely larger size should be adequate to detect presence of VPP in a herd. These calculations suggest that examination of 10 pigs from each herd, regardless of size, would be adequate to detect VPP as a herd infection.

The lungs from all pigs sampled should be entirely free of consolidation or discoloration. In herds which have previously been VPP-negative, lesions in any questionable lungs should be sampled for histopathologic examination. One VPP-positive animal should exclude all members of the herd for certification as VPP-free.

Discussion

The data indicate a high incidence of respiratory disease among swine (fig. 2). This is not a new observation and supports the observation made several years ago that abnormalities occurred in 60 to 70 per cent of the lungs in midwestern swine at market slaughter.[5] There are several relationships within the total area of pneumonias which should be discussed.

The apparent greater disagreement between microscopic and gross observation of VPP in October and November as compared with those in December, January, and February should not lead to the assumption that gross evaluation is unreliable. Management practices would conceivably bring groups of pigs together in the fall of the year that had not been together before. This could result in exposure of some of the pigs to VPP for the first time. Lesions of VPP develop slowly[2] so that pigs in the early stages of infection might appear

*This is a conservative figure since approximately 40 per cent of the total population is VPP-positive, which includes animals from positive and negative herds.

**We thank Dr. Charles Gardner for the statistics presented.

grossly normal yet have microscopic foci of infection. A history of management of herds inspected for certification as VPP-free would aid in the evaluation of the need of histopathologic assistance in diagnosis.

Another thing to be considered in certification of herds as VPP-free is the age of the pigs at the time of evaluation. Not all pigs in a VPP-infected herd have lesions or evidence of the disease; in general, these are the fast-growing, stronger pigs.[4] Slow growth may be associated with extensive VPP lesions. Selecting test lots from the average performing or poorer pigs would aid in diagnosis of VPP within a herd if the disease were present.

The suggestion that 10 animals be examined from each herd is intended to apply to detection of VPP only. Obviously, these same animals could be examined for atrophic rhinitis by observing cross-sections of their turbinates. No figures are available currently from which to determine what the sample size should be for atrophic rhinitis determinations. When such figures are available, the sample size should be set large enough to assure detection of either disease with a 99 per cent probability.

At the present time, we have no basis to judge the significance of the interstitial pneumonia observed in these studies. The lesions are similar to those in primary atypical pneumonia of man. They have not been observed in lungs of pigs in the swine repopulation project. This suggests the disease may be enzootic rather than epizootic in character although observation of "disease-free" herds for more seasons will be necessary to confirm this.

Summary

Lungs from pigs at market slaughter were examined for absence or presence of virus pig pneumonia (VPP). Lungs from 700 pigs were included which represented a total population of at least 3,500 pigs. Histopathologic examinations were made of tissues from lungs of 500 of these pigs.

In general, agreement between gross and microscopic diagnosis of VPP was satisfactory in lungs which showed typical gross lesions. Other respiratory diseases may be confused with VPP grossly, but generally not microscopically. Lungs that show any gross lesions should have tissue samples collected and sent to a laboratory for differential diagnosis.

Examination of 10 pigs from each herd, regardless of size, should be adequate to detect VPP as a herd infection.

Examination of lungs from 10 pigs from each herd regardless of its size is recommended for determining certification of a herd as VPP free. One VPP-positive pig among those examined disqualifies all members of the herd for certification as VPP-free because of the transmissible and persistent nature of the disease.

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Stress Conditions Important in Edema Disease

Hemolytic *Escherichia coli* found in association with edema disease in Canada are well distributed in normal pigs. Their presence in normal pigs may indicate that predisposing factors, such as changes in feeding, changes in housing, or nonspecific changes in environment, are still essential ingredients in pathogenesis of this condition.—*Vet. Rec.*, 71, (1959): 909.

Further Serologic Studies of

Brucellosis in White-Tailed Deer

of the Southeastern United States

Frank A. HAYES, D.V.M.
William T. GERARD, D.V.M.
Emmett B. SHOTTS, M.S.
Gloria J. DILLS

WHITE-TAILED deer (*Odocoileus virginianus*) are the major big-game animals in the southeastern United States, and it appears that within the next decade much of the suitable rangeland in this region will be reinhabited with this species.[1,7] Therefore, more information is needed on diseases which possibly are transmitted from these animals to domestic livestock. This particularly applies to brucellosis which is approaching complete eradication.[10,11]

Because of the dearth of information on this subject,[2,3,8] and the conceivable embarrassment it could cause the brucellosis eradication program, the Agricultural Research Service of the USDA recently has requested all available information on brucellosis in wildlife.[9] This directive also is significant for public health and many game conservation programs.[4,5] The following report represents a continuation of a regional study on brucellosis in deer of the Southeast.[12]

Collection and Serologic Procedures

Blood samples (2 to 8 ml.) were taken from deer on managed hunts or during trapping operations conducted in 1958-1960. "Kill-specimens" were collected in standard-type bleeding tubes after severing one of the major vessels; blood was procured by jugular puncture during trapping or restocking programs.

After collection, some specimens were refrigerated as long as 1 week before shipment to the laboratory for examination. Approximately 10 per cent of the samples were centrifuged shortly after collection and the serum was refrigerated and mailed to the laboratory within 6 weeks.

TABLE 1—Serologic Evidence of Brucellosis in White-Tailed Deer of Southeastern United States

	Total No. specimens	Serologic titers				
		1:25 (Nonspecific)	1:50 (Suspects)	1:100 (Reactors)	1:200 (Reactors)	1:400 (Reactors)
Alabama	905	5	2	2	1	1
Arkansas	558	1	1	1	0	0
Florida	494	2	1	1	1	1
Georgia	484	2	1	0	0	0
Kentucky	431	0	0	0	0	0
Louisiana	916	4	3	1	1	1
Maryland	422	3	3	1	0	0
Mississippi	334	1	0	0	0	0
South Carolina	45	1	1	0	0	0
Tennessee	864	1	1	0	0	0
Virginia	854	3	2	2	2	1
Totals	6,307	23	15	8	5	4

From the Southeastern Cooperative Deer Disease Study, Department of Pathology and Parasitology, School of Veterinary Medicine, University of Georgia, Athens.

On receipt, these specimens were centrifuged at 3,000 r.p.m. for 5 minutes, and standard plate agglutination procedures were used to determine the presence of Brucella agglutinins in the serum.[6] Initial testing was performed at an antigen: serum concentration of 1:25, and positive reactions were quantitated by 1:50, 1:100, 1:200, and 1:400 dilutions. Blood samples from 6,307 white-tailed deer were examined for significant *Brucella abortus* titers.

Results and Comments

The results of this survey are tabulated (table 1). If the serologic standards for testing cattle can be applied in evaluating tests with deer serum, 15 of the 6,307 animals, would be classified as "suspicious" (1:50), and only 8 as "reactors" (1:100). These data are in agreement with previous work and suggest that the white-tailed deer of the Southeast play an insignificant role as propagators of brucellosis in domestic animals.[12] The findings of this study also corroborate the results which were recently reported concerning the deer herds of Michigan.[13]

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ACKNOWLEDGEMENT

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A partial discussion of this study was presented before the 13th annual meeting of the Animal Disease Research Workers in the Southern States, North Carolina State College, Raleigh, April 2-3, 1959.

The authors thank the sportsmen, game biologists, rangers, and the P-R coordinators of the southeastern states for their wholehearted assistance in this project; also, Dr. C. J. Mikel, veterinarian-in-charge, Agricultural Research Service, Animal Disease Eradication Division, USDA, Atlanta, Ga.

Bacterial Antigens Irritate Udder Tissues

Antibody production was measured by agglutination tests on whey and blood serum after injection into the udder of the following antigens, alive or dead: *Brucella abortus, Salmonella pullorum, Staphylococcus aureus,* and *Trichomonas foetus.* It was possible to stimulate antibody production in the nonlactating udder by repeated infusions of dead antigen. No active antigen was found that did not irritate mammary tissue.—*Brit. Vet. J., 115, (1959): 105; abstr. 444 in Vet. Bull., 1960.*

Maduromycotic Mycetomas

in Animals—Brachycladium spiciferum Bainier as an Etiologic Agent

Charles H. BRIDGES, D.V.M., PH.D.
Joseph N. BEASLEY, D.V.M., M.S.

THE FOLLOWING is a report of mycetomas of the maduromycotic type in a cat, a horse, and a dog. A common etiologic agent is described for the cat and horse. These mycetomas are granulomatous lesions caused by infections with several of the higher fungi which usually form microcolonies in the tissue. The microcolonies, which are frequently pigmented, often can be seen with the unaided eye in the exudates and tissue. The disease was first reported in man.

In Madura, India, in 1842 and in 1846, two investigators described an unusual pathologic process in the foot of man, which was known locally as "Madura foot."[7,9] The fungal nature of this disease was established and the term "mycetoma" introduced in 1860.[4] Some forms contained pale yellow grains and others had black grains. In 1894, other investigators established that the black and yellow grains were of different character.[2] In 1913, these infections were described as true mycetomas and actinomycosis.[12] The true mycetomas were caused by higher fungi or Ascomycetes and Fungi Imperfecti, whereas actinomycosis then represented infection by the organisms now known as "Nocardia" and "Actinomyces."

The term "maduromycosis" was introduced for the true mycetomas and defined as "those forms of mycetoma with grains composed of large segmented mycelial filaments possessing well-defined walls, and usually chlamydospores."[6,12] Actinomycosis was defined as "those forms of mycetoma with grains composed of very fine nonsegmented mycelial filaments, in which usually the walls are not well defined from the contents, and in which chlamydospores are absent." Subsequent cases of maduromycotic mycetoma have been described in which hyphae were absent and the colonies were composed of chlamydospores.[16] The term "grain" used by these authors refers to the microcolonies of fungus which are found embedded in the granulomatous tissue and in the discharges from it.

The various organisms which have been identified as etiologic agents in man consist of several genera belonging to the Ascomycetes and Fungi Imperfecti, usually saprophytes in the soil or on plants.

Regardless of the great variety of genera and species involved in maduromycotic mycetoma, it must be recognized as a clinical and pathologic entity which takes a chronic course, usually of many months' and years' duration, being resistant to therapy other than surgical intervention which often must be drastic.

A mycetoma of the foot of a dog has been described in Alabama[14] and mycetomas on the foot of a horse, in the abdominal cavity of a dog, and on the feet of 2 other dogs have been described in Texas.[3] *Curvularia geniculata* (Tracy and Earle) Boedijn was isolated from lesions on 3 feet of 1 of the dogs.

The specimens obtained from the lymph node of a dog and from the nostril of a Turkish cavalry horse have the typical characteristics of mycetoma and both were pigmented.[1,13]

Allescheria boydii (Shear, 1921), a frequent etiologic agent in human mycetomas in the United States and other countries, has been isolated from a chronic eczematous lesion of a dog.[11] However, invasion of the dermis or subcutis was not present, the organism being restricted to the epidermis. To the authors' knowledge, this organism has not been isolated from mycetoma of animals.

Case 1

A chronic granulomatous enlargement of the foot of a 3-year-old female cat was diagnosed by histopathologic examination on May 29, 1958, as a mycetoma.* Subsequent treatment with various therapeutic agents failed to bring about recovery and the animal was referred to the authors for study.

When the cat was received, its foot was slightly enlarged and had several ulcerated areas from which a pink exudate could be

Dr. Bridges is a professor and Dr. Beasley an associate professor, Department of Veterinary Pathology, School of Veterinary Medicine, A. & M. College of Texas and Texas Agricultural Experiment Station, College Station.

*Submitted by Dr. J. A. Barrington, Wichita Falls, Texas.

expelled (fig. 1). Giemsa stains of this exudate revealed many clumps of nonpigmented mycelium with occasional interspersed chlamydospores, many within large foreign body giant cells (fig. 2). Numerous other short fragments were free in the exudate (fig. 3). Inoculation of 5 to 10 tubes of Sabouraud's agar with the exudate, on several occasions at intervals of a few days to several months, repeatedly resulted in the isolation of a fungus of characteristic morphology and growth.

A few weeks following the first expression of the exudate from the foot for purposes of culturing, the foot apparently healed and the animal was released to a new home. Approximately 3 months later, it was returned with a lesion which was larger than before. This resisted further treatment and the cat was euthanatized 1 year following the initial diagnosis.

Mycology.—Rapidly growing white mycelia appeared in 24 hours in the exudate which was inoculated on Sabouraud's agar and incubated at 20 C. It became gray-black in 48 hours. Elliptical, usually 3-

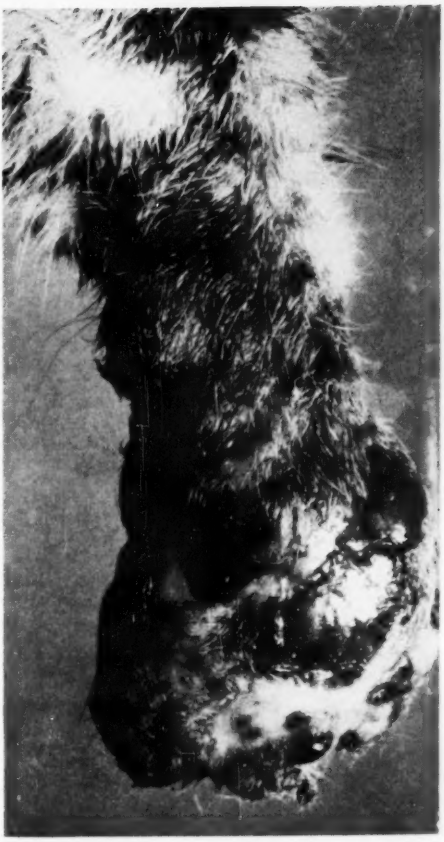


Fig. 1—Foot of cat showing enlargement and irregular tumefaction. x 1.1.

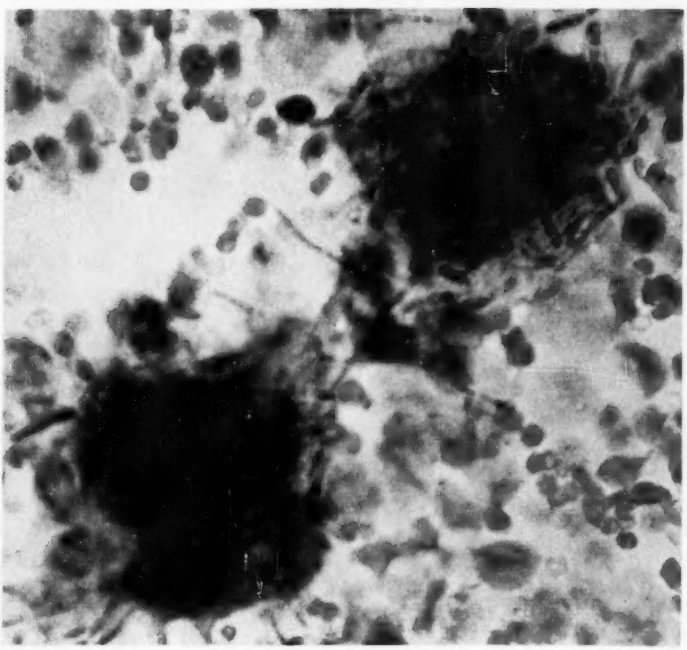
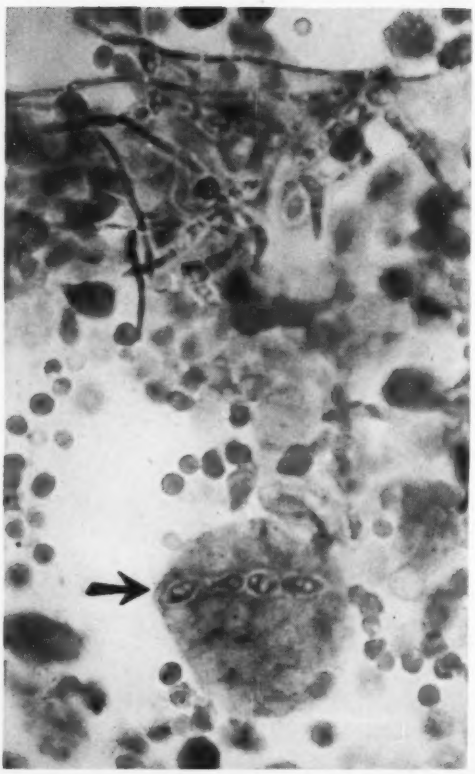


Fig. 2—Smear of exudate from cat's foot has the dense masses of mycelia. Giemsa's stain; x 350.



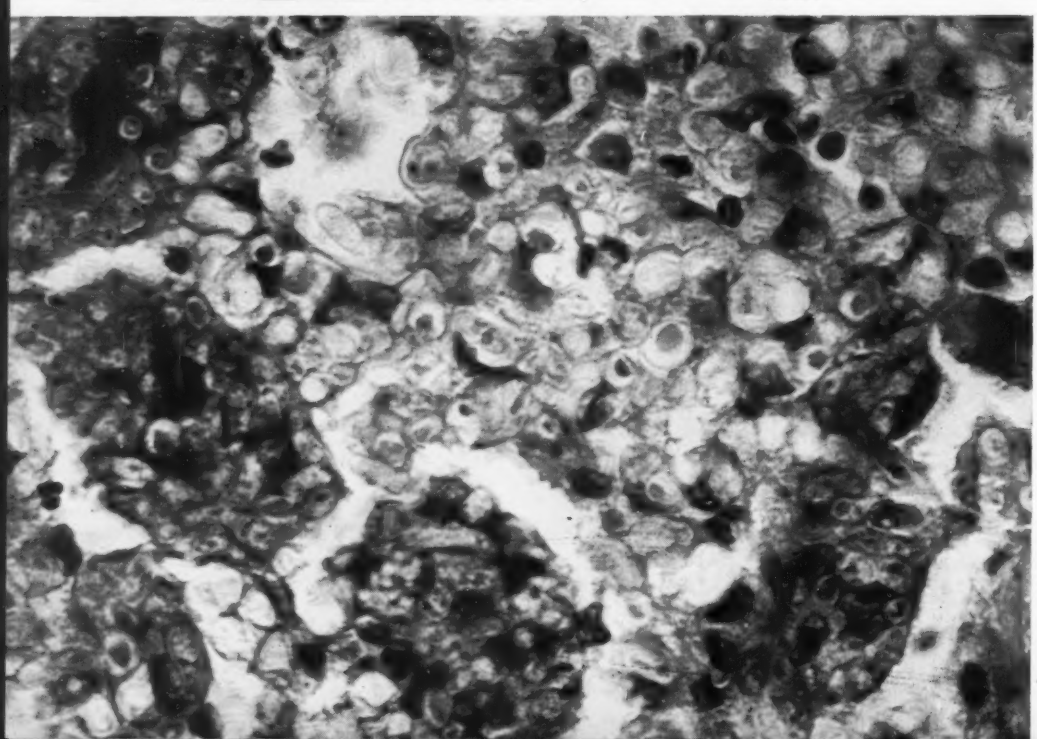
septate, elongate, thin-walled, dark brown to olive spores became apparent at about 96 hours postinoculation (fig. 7, 8). This organism was identified as *Brachycladium spiciferum* Bainier *Curvularia spicifera* (Bainier) Boedijn).*

Histopathology.—The lesion, which was largely subcutaneous, consisted of a rather uniform pattern of epithelioid tissue with numerous large foreign body giant cells scattered throughout. This granulomatous tissue was partially divided by occasional fibrous septa and contained numerous congested blood vessels which usually were surrounded by lymphocytes. A few neutrophils were scattered about and occasional microabscesses were present. The multinucleated giant cells frequently were sep-

*The organism was identified by Dr. Roderick Sprague, plant pathologist, Tree Fruit Experiment Station, State College of Washington, Wenatchee, Wash.; and Dr. J. E. Machacek, senior plant pathologist, Plant Pathology Laboratory, University of Manitoba, Winnipeg, Mani.

Fig. 3—Smear of exudate from cat's foot has scattered phagocytosed and free hyphae. Giant cell (arrow) contains small chlamydospores.

Fig. 4—Granulomatous tissue from cat's foot with several large giant cells containing many mycelia are visible in section with unstained wall surrounding central protoplasm. Nuclei of phagocytes are distorted by pressure of fungus. H & E stain; x 350.



arated by empty spaces from the adjacent tissue (fig. 4). Many cross sections of hyphae were present throughout, appearing as oval-to-round bodies representing internal cell constituents surrounded by clear areas of unstained fungus wall from the phagocytic cytoplasm. Staining by Gridley fungus technique[10] revealed dense masses of septate, branching hyphae and scattered chlamydospores present in the section (fig. 5). In the initial biopsy no pigmentation was evident in the fungus, but in the tissues removed at necropsy approximately 1 year later, many portions of the mycelia and numerous chlamydospores were dark brown (fig. 6). Chlamydospores were much more numerous in the tissue taken at necropsy.

Case 2

A 1-year-old horse with multiple small cutaneous and subcutaneous nodules approximately 6 to 8 mm. in diameter scattered over most of the body except the dorsum of the neck and back was presented for treatment. Mycetoma was diagnosed by histopathologic examination of several of the nodules.** The exact duration of the lesions was not known, but was considered to have been only a few weeks. The skin was movable over some but not all of the nodules, and a few had discharged exudate to the surface. Close examination of the cut surface of the excised lesions revealed many dark brown specks scattered through a pale pink tissue which was surrounded by a white fibrous capsule. Inoculation of the dark bodies on tubes of Sabouraud's agar and heart-brain infusion agar resulted in pure cultures of a grayish brown fungus. Microscopic examination of the cultures revealed white mycelia growing from them after 24 to 36 hours' incubation.

Mycology.—The fungus isolated from these lesions was similar to that from the cat except for a tendency for a few more of the spores to be slightly curved on their long axes. This organism also was identified as *Brachycladium spiciferum* Bainier (*Curvularia spicifera* (Bainier) Boedijn), the same as the organism isolated from the cat's foot (case 1).

Histopathology.—The nodular lesion consisted of a conglomeration of small granulomas with purulent centers in which were microcolonies of fungus consisting of heavily pigmented, thick-walled chlamydospores and a few nonpigmented septate hyphae (fig. 10). Numerous foreign body giant cells were scattered around the fungus and through the surrounding epithelioid cells (fig. 9). A few eosinophils were present in the fibrous connective tissue around the nodules, and lymphocytes were fairly numerous in the areas separating the small granulomas composing the nodules. Inhibition of the fungus by the tissue was indicated by the presence of ruptured and disintegrating chlamydospores, some phagocytosed by giant cells, as well as by the presence of a minimal amount of mycelium. In no lesion was there enough fungus to form the characteristic grains seen in the classical mycetoma.

Case 3

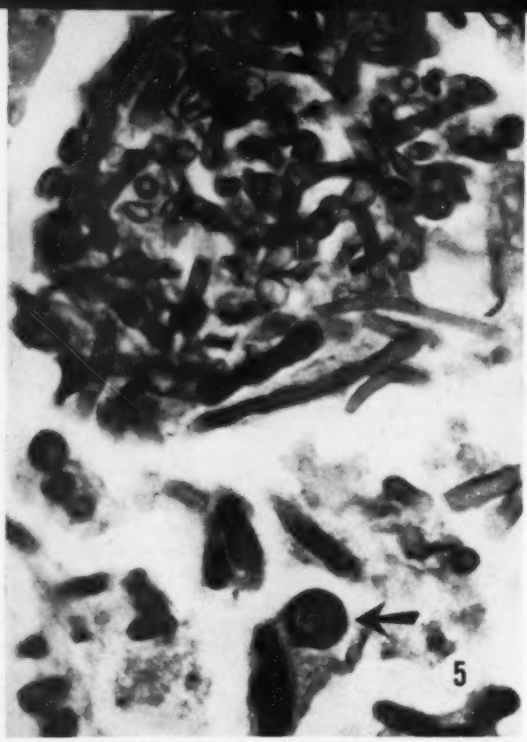
Prescapular lymph nodes on one side of an 8-year-old Shepherd-type dog had been enlarged more than a year. No other lesions were observed. The dog had been treated with sodium iodide which appeared to cause temporary regression of the enlargement. After euthanasia, the masses were excised and submitted as fixed tissues for histopathologic examination.† No similar lesions were found in the other tissues of the animal. Examination of the cut surface of the specimens revealed a granulomatous mass measuring approximately 6 by 4 by 5 cm. and surrounded by fascia and muscles (fig. 11).

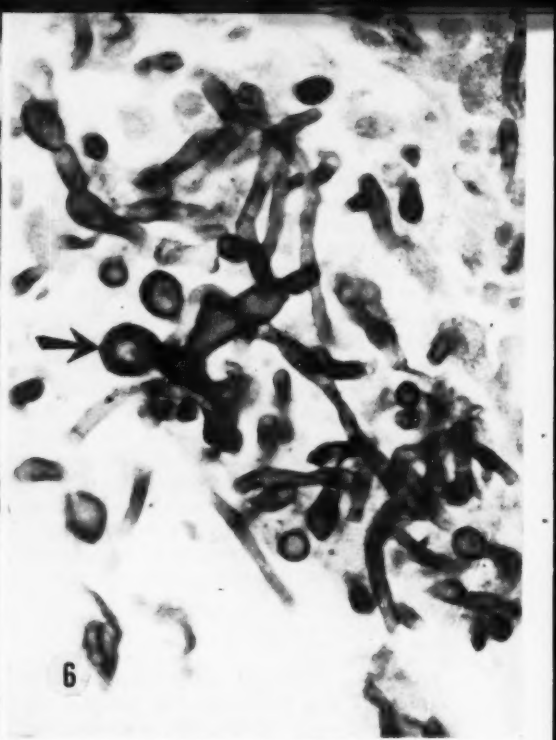
The granulomatous tissue was separated into smaller divisions by white fibrous septa. Scattered throughout the tissue were brown to black foci of granular material which were slightly more resistant to cutting than was the surrounding tissue. The pigmented material could be picked from the tissue with ease and was found to consist of small, irregular, occasionally vermiform microcolonies of fungus measuring 0.2 to 0.5 mm. in diameter (fig. 12).

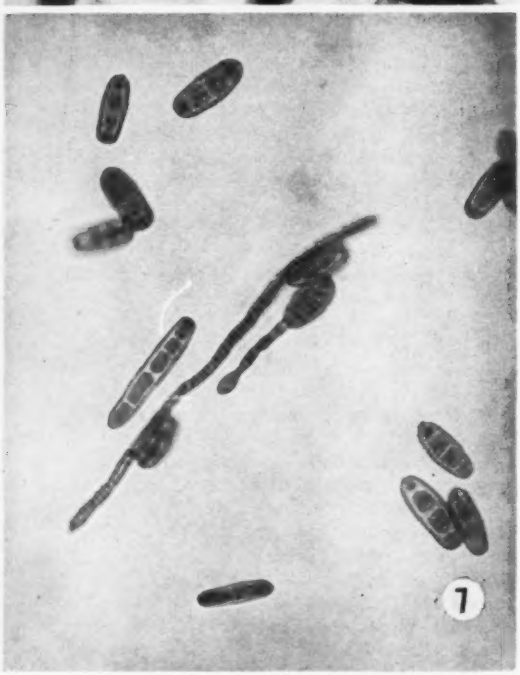
Histopathology.—The fibrous septa seen on gross examination separated foci of reticuloendothelial tissue in which were embedded 1 to several microcolonies of dark brown fungus. These consisted largely of thick-walled, pigmented chlamydospores

**Submitted by Dr. E. R. Willmann, Mason, Texas.

**Submitted by Dr. C. Tubbs, Cuero, Texas.







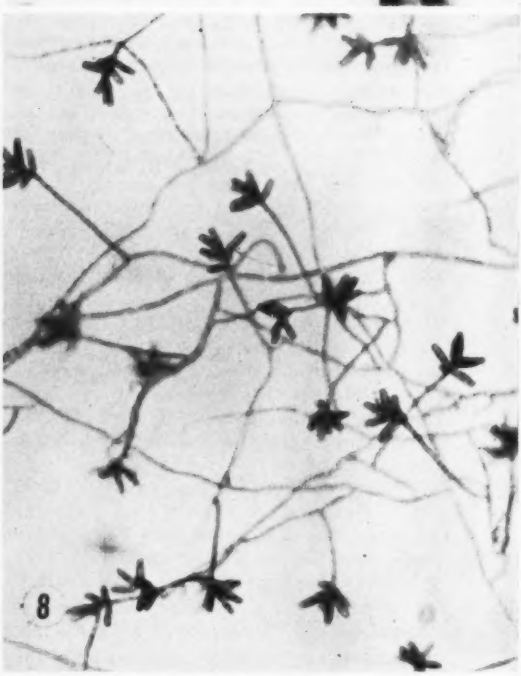


Fig. 5—Densely packed microcolony of fungus from cat's foot appears at top. Thick-walled pigmented chlamydospore is indicated by arrow. Gridley fungus stain; x 950.

Fig. 6—Heavily pigmented, thick-walled hyphae and chlamydospores (arrow) appear in granulomatous tissue of cat's foot. Gridley fungus stain; x 950.

Fig. 7—Spores from culture of Brachycladium spiciferum from cat's foot.

Fig. 8—Brachycladium spiciferum from cat's foot. Slide culture.

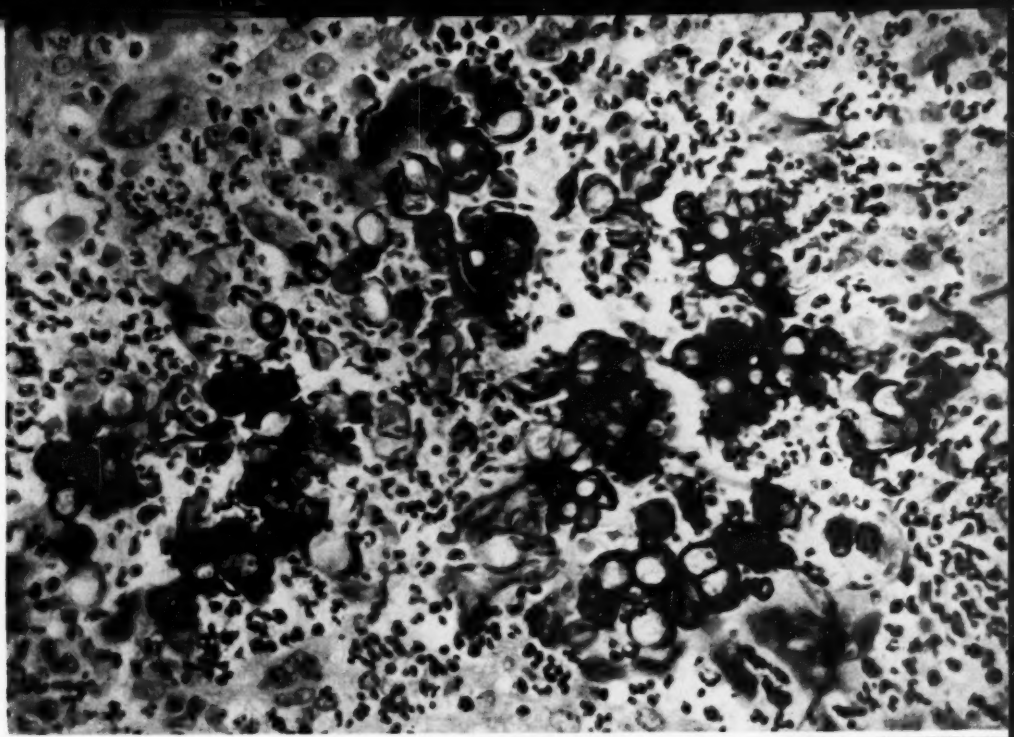


Fig. 9—Small microcolony of Brachycladium spiciferum in granulomatous lesion of horse's skin. H & E stain; x 533.

Fig. 10—Microcolony in lesion of horse's skin consisted largely of chlamydospores and a few fragments of septate hyphae (arrow). Gridley fungus stain; x 950.

arranged in thick lines which took horseshoe and scroll-like configuration (fig. 13). Much purulent exudate was present immediately around the microcolonies. On the inside of the convoluted masses of chlamydospores were septate, branching, pigmented and nonpigmented hyphae (fig. 14). These hyphae were most numerous adjacent to calcific deposits which were present within the encircled areas of the colonies, suggesting that here the organism was protected from the inhibitory elements of the host tissue. Degeneration of many chlamydospores was indicated by the presence of numerous empty ruptured forms scattered throughout the microcolonies.

Discussion

These cases of mycetoma vary somewhat in gross and microscopic characteristics. At the time of the original biopsy of the lesion on the cat's foot, the fungus consisted of nonpigmented mycelia with less tendency to be restricted to discrete

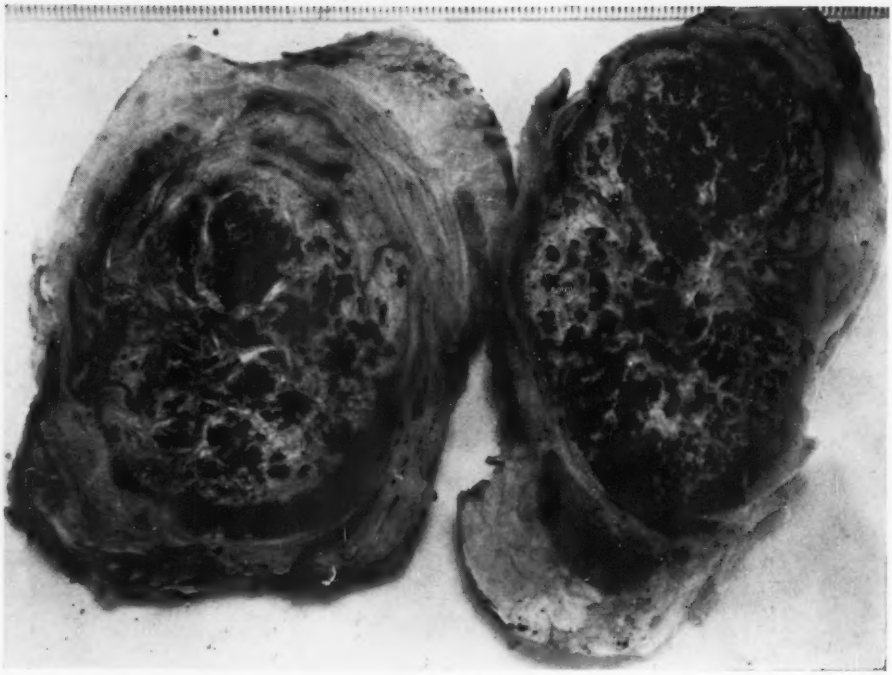


Fig. 11—Cut surface of prescapular lymph nodes of dog has masses of heavily pigmented microcolonies of fungus embedded in granulomatous tissue. Muscle and fascia surround the masses. x 1.0.

Fig. 12—Microcolonies of fungus dissected from dog's foot (case 3). x 20.

microcolonies than those seen in the more classical form such as that of the dog. However, a few definite chlamydospores were scattered at random, and transitions from diffuse infiltrations of mycelium to discrete microcolonies were present (fig. 2, 5). A few months later, the lesion on the foot had grown larger and at this time pigmented mycelia and chlamydospores were quite evident.

On the other hand, the lesions in the skin of the horse were of relatively short duration and during examinations were small and confined. All nodules which were not excised, healed spontaneously within 3 months following diagnosis. The fungus consisted largely of heavily pigmented chlamydospores and occasional nonpigmented hyphae. These focal collections of fungal elements were approximately the same over-all size as the width of those

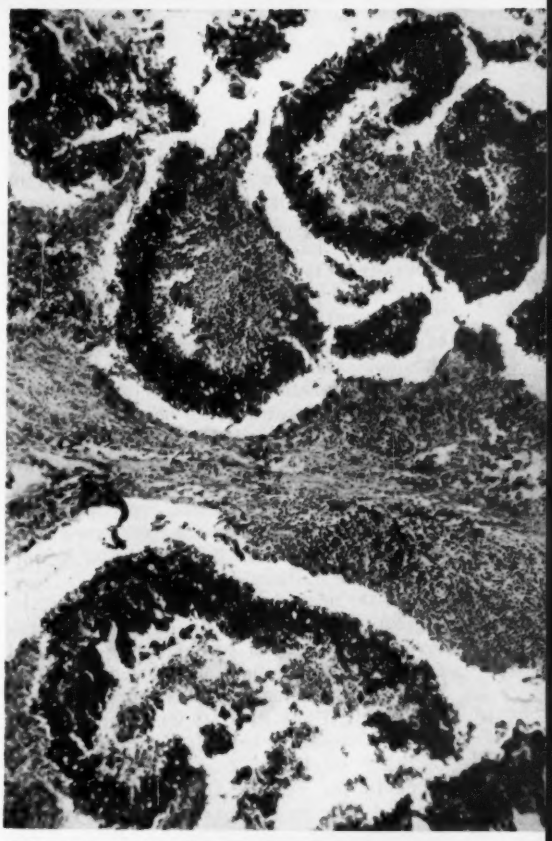


Fig. 13 (right)—Scroll-like configuration of cross sections of microcolonies such as seen in figure 12 as they appear in tissue sections. H & E stain; x 75.

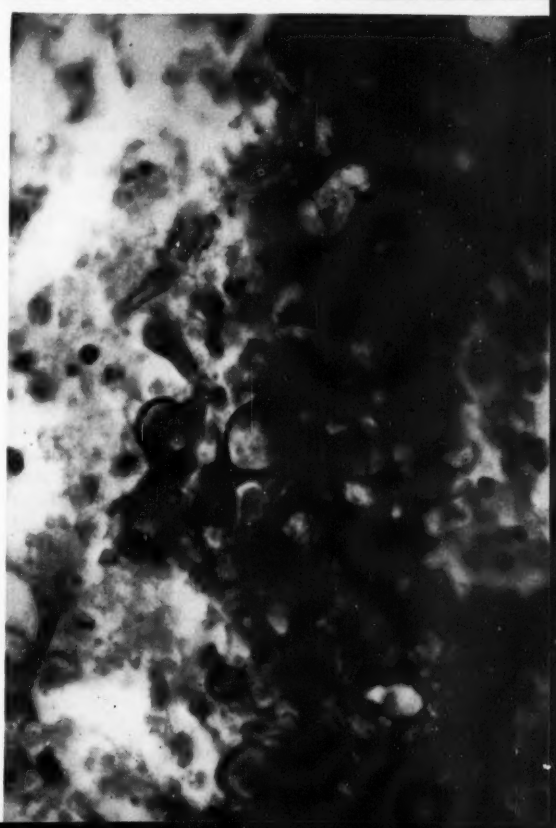


Fig. 14 (below, right)—Higher magnification of microcolony from figure 13 shows hyphae at inner edge of the zone of pigmented chlamydospores. H & E stain; x 740.

zones of chlamydospores in the more classical microcolonies of the dog.

Thus, it appears that the fungus in the cat's foot was not sufficiently inhibited by the host tissues to require formation of any large number of resistant chlamydospores. The fungus in the horse's skin was inhibited sufficiently that it was represented largely by resistant chlamydospores. Whereas ruptured and degenerating chlamydospores were frequently seen in the horse, none were positively identified in the cat.

The small size of the microcolonies in the horse is possibly related to the short duration of the infection as well as to inhibitory effects of the host's tissue. Other mycetomas in horses previously reported have had large colonies resembling those of the dog's foot described here although the mycelial component has been minimal.[1,3] This indicates that the variation in the relative arrangement and composition found in the microcolonies in the tissues is dependent upon the delicate balance of aggressiveness and resistance between the parasite and the host. Certainly the variable in these cases could exist in both the infecting organism and in the host.

The development of hyphae adjacent to the calcific deposits in the exudate about the colonies of the dog where they might be partially protected is suggestive of such a delicate balance.

A thorough understanding of the possible variation in morphologic forms of microcolonies seen in these mycetomas should make it possible to identify other mycetomas in which the fungus is not in classical colonies such as represented (case 3). It appears that the horseshoe and scroll-like forms are only 1 colony type which may be present and transitions between all phases may be seen in some mycetomas. The basic features appear to be the occurrence of chlamydospores with or without mycelia in typical lesions. The occurrence of pigment is additional evidence of the character of the organism. However, case

1 emphasizes an earlier report that the same fungus can produce pigmented or nonpigmented forms in the tissue.[12] A combination of chlamydospores and hyphae, especially with pigmentation is not usual with other types of granuloma-producing mycotic infections.

One must differentiate the microcolonies of maduromycotic mycetomas from that of chromoblastomycosis, a somewhat similar lesion containing pigmented fungus but lacking hyphae and being characterized by thick-walled pigmented cells with internal cross walls. The fungus in this latter pathologic entity may resemble that of maduromycotic mycetoma with minimal or no mycelium when large colonies are formed. No authentic cases have been reported in animals to the authors' knowledge.

The lesions in the dog and cat had the typical clinical behavior of the maduromycotic mycetomas in that they persisted for a long time, fluctuating under the effects of treatment but being resistant to treatment. They grew to considerable size, yet the causative organism did not spread to other localities. The multiplicity of lesions in the skin of the horse can be explained only on the basis of multiple inoculations. The spontaneous healing of the lesions in this horse suggests the possibility that such less persistent infections might occur more frequently than is recognized.

Microcolonies escaped from sinuses to the surface of the skin in 2 of the cases but not from the lesion in the dog's lymph nodes because of the lack of invasion of the overlying skin and formation of fistulas. The variation in the character of the colonies reflects the stage of infection and the host-parasite relationship.

There appears to be a close taxonomic relation of the fungi which have been isolated from maduromycotic mycetomas in animals. *Curvularia geniculata* (Tracy and Earle) Boedijn which was isolated from multiple mycetomas[3] in a dog is a recognized saprophyte and occasional parasite on certain small grains and grasses. Similarly, *Brachycladium spiciferum* Bainier (*Curvularia spicifera* (Bainier) Boedijn) is a fungus which is isolated from overwintering roots and crowns of perennial grasses, and from roots and sprouting seeds of grasses and cereals in early season and late fall. It is quite similar to the genus Curvularia to have been classified in this group.[15] Also, *Brachycladium spiciferum* appears to be closely related to *Helminthosporium tetramera.** A species of Helminthosporium has been isolated from a mycetoma in a dog.[8]

One must consider the possibility of misinterpreting the significance of the growth of these organisms on inoculated mediums because such organisms may be frequent contaminants of bacteriologic mediums.[14] However, 23 pure cultures of the organism were obtained from the cut surface of surgically removed lesions in the horse, using aseptic technique. None contained other fungi and only 1 was sterile. Examination of this tube of medium by dissecting microscope revealed that none of the pigmented microcolonies had been deposited on it. In the other tubes of mediums from which isolations were made, colonies of the organism could be seen to arise from pigmented microcolonies deposited on the medium by inoculation of infectious material. Similar results were obtained in over 40 tubes of mediums inoculated with exudate obtained from the cat's foot under aseptic conditions on 5 occasions during a period of approximately 1 year.

The question of terminology for this clinical and pathologic entity is not an infrequent subject of consideration. As pointed out in the introduction, the characterization of the condition has developed through a series of stages, each introduced by the advent of additional knowledge or insight. Although maduromycosis is the most commonly accepted term in world literature, some investigators prefer the original name "mycetoma" to "maduromycosis" because the latter designates infections caused by several unrelated species in some 8 or 10 genera of Hyphomycetes and Ascomycetes.[15] The name can be a source of confusion by its derivation from a geographical site to which the disease is not limited; by derivation it suggests both the genus Madurella which is involved in some mycetomas, and *Nocardia madurae* which is also a cause of mycetoma but not indicated under the term "maduromycosis." Certainly the use of "madura foot" is not indicated because a good percentage of the cases involve sites other than the foot, e.g., abdominal cavity, cervical, thoracic and abdominal skin, nasal mucosa, and pre-

*Drs. Sprague and Machacek, who identified the organisms in this report, consider Brachycladium spiciferum Bainier closely related to Helminthosporium tetramera.

scapular lymph nodes. The authors' have designated the condition as "maduromycotic mycetoma."

Summary

Maduromycotic mycetomas have been diagnosed in a cat, a horse, and a dog. *Brachycladium spiciferum* Bainier was identified as a causative agent in the cat and the horse.

Generally, the clinical signs were manifested by chronic inflammation with formation of nodular granulomatous masses in the foot of the cat, skin of the head and body of the horse, and in a prescapular lymph node of the dog. Pigmented colonies of fungus could be seen as brown to black specks in the lesions from the dog and the horse. Colonies of fungus were easily found in stained smears of pus taken from draining sinuses of the cat's foot.

Although there was considerable variation in the shape of the microcolonies in all 3 animals and in the pigmentation of the fungus in the cat's foot from time to time, chlamydospores and hyphae were present at all times.

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Carcinogenicity of an Iron-Dextran Complex

Iron-dextran complex was markedly carcinogenic in the rat and mouse after subcutaneous injections of massive doses. Most of the tumors appeared at the site of injection as sarcomas arising from the iron-laden connective and reticuloendothelial tissues.

Dextran was inactive. It is not known if the carcinogenic effect is a function of the iron alone or of the entire iron-dextran complex.

In experiments designed to test successively lower dosages, 1 sarcoma appeared after 8 months in a series of mice receiving weekly subcutaneous injections of 0.05 cc. of iron-dextran complex.—*Nat'l. Cancer Inst. J., 24, (1960): 109; abstr. 40,422, the Upjohn Co.*

Editorial

On Purchasing Drugs and Biological Products

Steadily rising costs in practice have forced many veterinarians to become exceedingly price conscious where drugs and biological products are concerned. Product expenses that approach 40 to 50 per cent of gross income are not uncommon and are substantial. But reducing drug expenditures is not a simple matter. Cutting drug expenses by buying lower-priced products often results in acquisition of lower quality products. On the other hand, buying higher-priced products does not always assure the purchaser of getting the best product. Because they are seldom able to determine the comparative quality of products, many veterinarians have concluded that quality of products is similar and that price is the most important factor to consider.

Product Quality Differs

But quality of all products is not the same. Chicken embryo origin (CEO) rabies vaccines of several manufacturers recently were tested at a state laboratory, and only one brand was found to be sufficiently potent to give the protection expected by the purchaser. Recognizing the danger revealed by these tests, the ARS immediately imposed more rigid testing and inspection procedures to assure adequate potency of products (*see J.A.V.M.A.*, June 1, 1960: 538). But, up to this time, many had thought all CEO rabies vaccines were the same, except in price.

Not many months ago, another laboratory found that CEO distemper vaccines of many manufacturers were not stimulating adequate immunity in dogs. Judging by titration of blood serums, some vaccines afforded no protection at all, while others induced a high level of immunity. Yet, all these products sold at about the same price. Obviously, they were not the same in quality.

Pharmaceutical products are not always similar in quality either. A number of veterinarians have been surprised to find, on rumenotomy or necropsy, undissolved medicated boluses in the rumen of a cow to which the boluses had been given a week earlier. Obviously, a readily dissolving bolus would have been a superior product.

Why Similarly Produced Products Differ

Why should there be differences in products that supposedly are produced and inspected under similar circumstances? Reasons are several.

Handling.—Live virus products that have passed inspection at the manufacturing plant may have lost potency by the time they are actually used by a veterinarian at his hospital or on the farm. This loss may be due to careless or otherwise improper handling during storage or shipment. Products requiring refrigeration, such as modified live virus vaccines, cannot be expected to retain a full measure of potency if they are hauled long distances in unrefrigerated trucks or arrive at the veterinarian's office with preservative ice melted, especially in hot weather. Handling of products may vary from company to company, within companies, and among individual users.

Quality Control.—Sometimes products pass inspection immediately following production, but they change or deteriorate as time passes. Sometimes this change is manifested as precipitation which makes a product for intravenous use dangerous. Sometimes changes result from contamination with molds, which does not become apparent until weeks have passed. Sometimes consistency of the preparation changes so that a product may become difficult to expel from a tube, or it may become thick and chunky in solution, making it unhandy to use. Sometimes products undergo inapparent chemical changes that cause irritation, urticaria, or shock. The fact that these changes do occur in some products, if only occasionally, strongly indicates that quality of products of all manufacturers is not the same. The conclusion that must be drawn is that some manufacturers maintain better control of product quality than others do.

Improper Testing and Reporting.—On occasion, a product reaches the market without being tested properly. In these cases, proof of a product's value may have been based mainly on biased or limited reports rather than on well-designed and conducted scientific tests.

Sometimes manufacturers cull unfavorable reports with the result that only favor-

able ones are published. This culling technique prevents the buyer from learning the true worth of the product, except by trial at his own expense. In time, deliberately one-sided reporting becomes apparent when highly touted products fail to give expected results under actual practice conditions.

Even regulatory agencies, such as ARS and FDA, must depend on manufacturers' submitted reports. The agencies themselves have extremely limited facilities for testing products. Consequently, it is the integrity of the manuacturer rather than the government agency that determines whether a product is adequately and properly tested before marketing.

Limitations of Government Control

Inspection by a government agency does not assure the veterinarian that products will always possess attributes that he expects them to have. Government regulations set only certain minimum standards. Often they do not require determination of vital information, such as duration of immunity provided by a vaccine (there is no requirement for manufacturers of *Leptospira pomona* bacterin to show how long immunity will last), dissolving time for boluses or tablets in various species, or irritative factors developed in a drug several months after bottling.

Potency and sterility are usually determined in small numbers of laboratory animals. Many tests do not require that potency be determined in animals destined to be given the products. For example, the ARS does not insist that CEO distemper vaccine potency be determined in pups. *Leptospira pomona* bacterin potency is determined in hamsters and guinea pigs, not cattle and swine. The effect in the laboratory animal is a reflection of potency in the farm or pet animal concerned, of course, but it has obvious limitations. Duration of immunity produced in the veterinarian's patients cannot be readily determined from the duration of immunity in laboratory animals.

Intelligent Purchasing

There is no simple solution to the problem of intelligent product purchasing. The buyer's only protection is knowledge of each product. To acquire this knowledge, he should insist on complete descriptive literature of all products he intends to buy. If the manufacturer can't supply literature, it is logical to assume that the manufacturer doesn't have the facts the buyer needs. Word-of-mouth assurances from detail men are poor substitutes and may have little or no basis. A proper evaluation of a drug can be made only by analyzing tests scientifically conducted on fairly large numbers of animals by independent investigators. If the manufacturer cannot supply this information about a product, the veterinarian must assume that the product has not been adequately tested and its purchase must be considered a gamble.—A. F.

What Physicians Should Know About Drugs

Every physician should have the following information concerning a drug: (1) the names by which the drug is known, together with a full exposition of its chemical constitution and make-up; (2) how the drug can easily be identified; (3) its physical and chemical properties; (4) data on absorption, distribution, metabolism, and removal of the drug from living material with particular reference to the rates; (5) local action at point of application; (6) systemic action upon particular body organs and details of activities at the cellular level on enzyme systems and on metabolic cycles; (7) toxicity in single or repeated doses, with symptoms and specific data on terms of time-concentration relations and methods of controlling toxicity; (8) recommended uses; (9) data on controlled clinical use with analysis of effectiveness and details of untoward reactions; (10) methods of administration with recommended dosage in terms of mass of drug per mass of living material; and (11) preparations available.—*C. D. Leake, Mod. Med., 28, (May 15, 1960): 14.*

from the *Research Journal*

Serotypes of Avian PPLO

The antigenic relationships of avian pleuropneumonia-like organisms (PPLO) were determined by agglutination, agglutinin absorption, and hemagglutination-inhibition reactions in rabbit antiserums prepared against 15 isolates obtained from various avian and geographic sources. Eight serotypes were identified, of which only 1, serotype A, contained isolates capable of producing lesions and readily detectable serologic titers in turkey poults; *i.e.*, so-called "pathogenic PPLO." Certain other biological characteristics were correlated with pathogenicity or nonpathogenicity; strains in the pathogenic serotype were consistently slower in growing, yielding forms apparently similar to Nelson's coccobacilliform bodies and produced small (up to 0.1 mm. in diameter), refractile, granular, raised, and uniformly stained colonies. This was in contrast to "ring forms" observed in the nonpathogenic serotypes, where the colonies were larger (0.3 to 0.5 mm. in diameter), had raised centers, and showed a dark center and lacey periphery when stained. Only the strains in the pathogenic serotype possessed ability to hemagglutinate chicken erythrocytes.

Pathogenic and nonpathogenic PPLO could not be differentiated on the basis of carbohydrate fermentation since 4 of the types, including the pathogenic type, actively fermented carbohydrates; however, 4 of the nonpathogenic types failed to ferment carbohydrates. — [*Albert L. Kleckner: Serotypes of Avian Pleuropneumonia-like Organisms. Am. J. Vet. Res., 21, (March, 1960): 274-280.*]

Cerebrospinal Fluid Electrolytes in the Dog

This report is the result of an investigation to determine normal electrolyte values for canine cerebrospinal fluid. Aspiration of cerebrospinal fluid was performed on anesthetized dogs by puncture of the cisterna magnum with a 20-gauge, 1½-inch needle. A total of 129 samples of cerebrospinal fluid was taken from 83 dogs of mixed breeding and sex. Sodium and potassium concentrations were determined on a Beckman DU flame spectrophotometer. A Cotlove chloridometer was used for chloride determinations. Inorganic phosphate and calcium were determined on an autoanalyzer. The following mean values were obtained: Na, 156.3 mEq/1; K, 3.1 mEq/1; Ca, 6.2 mg./100 ml.; Cl, 125.5 mEq/1; inorganic phosphate, 1.1 mg./100 ml.—[*Roger A. Yeary, Ira L. Shannon, and John R. Prigmore: Cerebrospinal Fluid Electrolytes in the Dog. Am. J. Vet. Res., 21, (March, 1960): 306-307.*]

Larvacides for Ascaris in Pigs

Ronnel and Bayer 21/199 (organic phosphorothioates) were fed to baby pigs for 5 days at 50 and 12.5 mg. per kilogram of body weight, respectively. This level of medication was toxic to the host but had no effect on the migrating larvae of *Ascaris suum*.—[*G. W. Kelley and C. L. Marsh: Lack of Larvacidal Action of Ronnel and Bayer 21/199 Against Migrating Ascaris suum in Baby Pigs. Am. J. Vet. Res., 21, (Jan., 1960): 109-110.*]

Agglutination Test for Pseudotuberculosis

Pseudotuberculosis is a common disease in Sudan and most of the information on its frequency comes from meat inspection statistics. Various serologic tests were used to diagnose the disease in the living animal, but the main difficulty in perfecting these tests was the tendency of various strains of *Corynebacterium ovis* to clump. By studying the

clumping rates of different strains, it was possible to find a strain suitable for preparing a bacterial suspension for the agglutination tests. This suspension was a 5 per cent salt solution containing 10 to 20 per cent heated normal rabbit serum with the pH adjusted to 6.8.

The agglutination tests were found to be specific in the diagnosis of pseudotuberculosis in sheep; the slide test was preferable to the tube test. — [*F. I. Awad: Serologic Investigation of Pseudotuberculosis in Sheep. I. Agglutination Test. Am. J. Vet. Res., 21, (March, 1960): 251-253.*]

New Books

Symposium on Basic Research

This review cannot abstract all of the 15 presentations, each of which provides stimulating as well as sobering reading. The opening paper by J. Robert Oppenheimer on the "Need for Newer Knowledge" has as its theme that support for basic research is needed because knowledge is useful and the search for it is enobling. Dr. Oppenheimer found it hard to think of any advances in science which have not had large practical consequences. Alan T. Waterman of the National Science Foundation made clear that the borderline between basic and applied research is not distinct. He also said that the potential of this country in science is not being realized and that there must be more support for basic research.

Speaking of basic research in liberal arts college, Laurence M. Gould, president of Carleton College, concluded that an educational program is needed for college administrators and trustees to awaken them to the importance of fundamental research in the teaching process. Dr. Gould also emphasized the need for support of undergraduate research in order to recognize talent early and to encourage it.

Discussions on basic research in universities by C. A. Elvehjem and L. A. DuBridge were devoted largely to financing such research and the problems arising from governmental support. Of particular concern to Dr. DuBridge is the failure of governmental grants to pay full cost of sponsored projects. This requires that universities must find other funds to make up the difference in cost. Dr. Elvejhem believes that the greatest expansion of fundamental research should be in universities because of the greater opportunities for freedom in investigation. M. A. Tuve of the Carnegie Foundation proposed that public funds be used to support a creative investigator for a lifetime. Such support would include payment for technical assistants and students. Dr. Tuve stated that for the cost of $40 to 60 million we would have, in 10 years, a solid phalanx of 500 to 600 outstanding investigators free to devote their time to creative ideas for their lifetime. Paul E. Klopsteg of the National Science Foundation discussed a proposal to amend the internal revenue act which would make it more attractive for people in all income brackets to make contributions to institutions of higher learning. This subject elicited more questions from the audience than any other proposal for improving support of basic research.

Papers by participants from industry, educational institutions, and government were in accord with the idea that fundamental research is urgently needed and must be supported to a greater extent. C. H. Greenewalt of E. I. du Pont de Nemours and Co. called basic research a technologic savings account from which we make withdrawals for applied research. To guard against bankruptcy of ideas, it is necessary that support of basic research replenish the account.— [*Symposium on Basic Research. Edited by Dael Wolfle. 328 pages. American Association for the Advancement of Science. Publication No. 56. Washington, D. C. 1959. Price $3.00; $2.50 to AAAS members.*]—ALFRED G. KARLSON.

News

Veterinary Team Receives USDA Superior Service Award

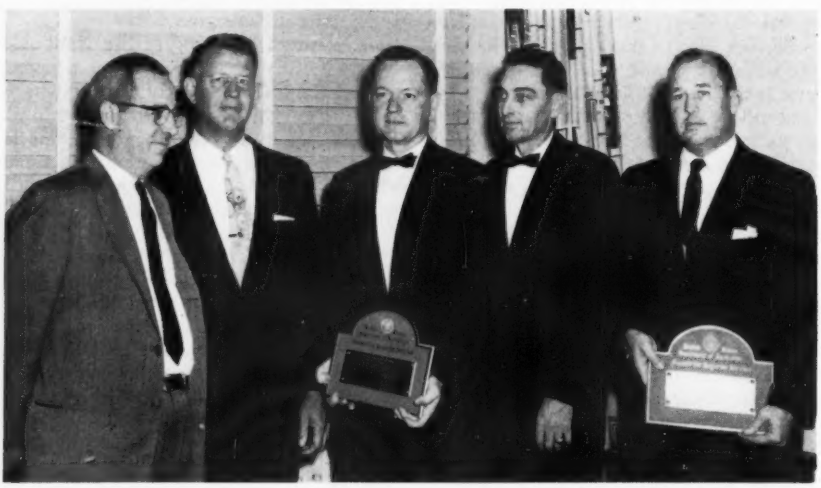
A four-man team of federal veterinarians who developed training programs for agricultural personnel charged with protection of the nation's meat and livestock against radioactive contamination have been presented a unit Superior Service Award by the USDA.

The team members are: Dr. James D. Lane, director of the Department's Meat Hygiene Training Center in Chicago; Dr. Robert P. McCoy, Jr., veterinary meat inspector, Washington, D.C.; Dr. Robert A. Moody, supervisory veterinary inspector, Nampa, Idaho; and Dr. Ted Rea, supervisory veterinary livestock inspector, Austin, Texas.

In 1957, these men were selected to develop and present training programs in radiological defense as part of the USDA's responsibilities in civil defense activities. To better understand the handling of radioactive contamination, the team was assigned to the Atomic Energy Commission to take part in a fallout study in Nevada, in 1957, which involved detonation of 18 atomic bombs and six months of study of radioactivity in the desert. Through individual study, they gained background in the physical sciences, radiological instrumentation, radiobiology, and related subjects.

During the past 18 months, they have, in turn, trained more than 400 key USDA personnel. Trainees included livestock regulatory officials having nation-wide responsibilities for the health of livestock and the wholesomeness of food products derived from animals and many other Department officials responsible for making policy decisions in the field of agriculture.

The team was nominated for the Superior Service Award because of their unusual devotion, great personal sacrifice, and high measure of success in presenting the training courses. The award was presented by Dr.



The USDA Superior Service Award was presented to a four-man team of Department veterinarians shown here with Dr. Byron Shaw (left), administrator of ARS. The group is responsible for the development of training programs in radiological defense in agriculture for key agricultural personnel.

Left to right—Dr. Shaw who presented the Award; Dr. Robert Moody, meat inspector in charge for MID, Nampa, Idaho; Dr. James D. Lane, in charge of MID's Meat Hygiene Training Center, Chicago; Dr. R. W. McCoy, staff officer for Labels and Standards of Identity for MID; Dr. Ted Rea, assistant veterinarian in charge in Texas for the Animal Disease Eradication Division in Austin.

Byron Shaw, administrator of the Department's Agricultural Research Service.

Dr. Robert Moody (OSU '38) is a native of Columbus, Ohio. He began work with USDA's Brucellosis Eradication Force in 1938 and transferred to the Meat Inspection Division (MID) in 1939. During World War II, he served as an Army veterinary officer in New Guinea and Australia. After the war, he served with the MID in Fresno, and San Francisco, Calif., and was made inspector in charge of Nampa, Idaho, in 1959.

Dr. Ted Rea (TEX '43) was born in Weatherford, Texas. He served with USDA's cooperative foot-and-mouth disease eradication program in Mexico for about five years. He also served with the Animal Disease Eradication Division in Phoenix, Ariz., and in Albuquerque, N. M. He was recently promoted to the position of assistant veterinarian in charge in Texas, with headquarters in Austin.

Dr. James D. Lane was born in Columbus, Ohio, and received his D.V.M. degree from Iowa State University in 1943. He entered the Department's Meat Inspection Division in 1948 at Oklahoma City and later served at South St. Paul, Minn., and at Memphis, Tenn. In 1959, he was transferred to the Division's procedures and training office Washington, D.C., and in January, 1960, became head of the Meat Hygiene Training Center in Chicago, Ill.

Dr. R. W. McCoy was born in Port Arthur, Texas, and received his D.V.M. degree from the A. and M. College of Texas. He served during World War II as a Navy fighter pilot and was in private veterinary practice for about five years. He entered the meat inspection service in Denver, and later served in Boise, Idaho, Purcellville, Va., and New York City. He is now a staff officer for Labels and Standards of Identity, MID, in Washington, D.C.

Parasitologists' Association to Hold Symposium on Anthelmintic Research in Denver

The fifth annual meeting of the American Association of Veterinary Parasitologists will be held at the Denver-Hilton Hotel in the Denver Room, from 9 a.m. until 5:30 p.m., on Sunday, August 14, the day preceding the opening of the Ninety-Seventh Annual Meeting of the AVMA.

The program will be devoted to methodology in antiparasitic research. Qualified investigators from universities, governmental agencies, and industry will speak on several aspects of development, testing, and clearance of chemical agents for parasitic control; equivalent time will also be allotted for discussion of each topic.

All interested persons are cordially invited to attend and to participate.

s/F. R. Koutz, *Chairman.*

Dr. L. O. Mott to Head Virological Investigations at NADL

Beltsville, Md.—Dr. Lawrence O. Mott (KSU '29) has recently been designated as research leader of the virological investigations group at the National Animal Disease Laboratory, Animal Disease and Parasite

Dr. Lawrence O. Mott

Research Division, ARS, USDA. Upon completion of the new Animal Disease Laboratory at Ames, Iowa, Dr. Mott's research activities will be transferred there from Beltsville.

Recognized as an authority on viral disease of domestic animals, it is due to Dr. Mott that a highly accurate diagnostic test for anaplasmosis has been developed which is currently being used in various state laboratories. He has been connected with the Department of Agriculture since 1937.

Dr. Mott was also instrumental in developing procedures for the production of a vaccine for foot-and-mouth disease which was used to eradicate this disease from Mexico.

S/EDWIN R. GOODE, JR., *Assistant Director.*

Schedule of Laboratory Refresher Training Courses

Following is a list of the laboratory refresher training courses which are to be offered by the Laboratory Branch of the Communicable Disease Center, Atlanta, Ga., from Oct. 10, 1960, to April 7, 1961:

Fundamentals of virology—Oct. 10-12, 1960
Laboratory methods in the diagnosis of TB—Oct 31-Nov. 11
Laboratory methods in the diagnosis of rabies—Nov. 28-Dec. 2
Bacteriophage typing of staphylococci—Dec. 5-9
Laboratory methods in medical mycology—Jan. 9-Feb. 3, 1961
Serologic methods in microbiology—Jan. 23-Feb. 10
Laboratory methods in the diagnosis of TB—Jan. 30-Feb. 10
Laboratory methods in the study of pulmonary mycoses—Feb. 13-24
Laboratory methods in medical bacteriology—Feb. 27-March 17
Laboratory diagnostic methods in veterinary mycology—March 6-10
Laboratory methods in the diagnosis of viral rickettsial diseases—March 13-31
Special problems in medical bacteriology—March 20-24
Laboratory methods in enteric bacteriology—March 27-April 7
Laboratory methods in the diagnosis of rabies—April 3-7

The following courses are given by special arrangement only:

Laboratory methods in the diagnosis of malaria—1 week
Special training in virus techniques—2-4 weeks
Typing of Corynebacterium Diphtheriae—1 week
Special problems in enteric bacteriology—2 weeks
Phage typing of Salmonella Typhosa—1 week
Laboratory methods in the diagnosis of leptospirosis—1-4 weeks
Serologic differentiation of streptococci—2 weeks
Special problems in microbiology—1-2 weeks

Information and application forms may be obtained from the Laboratory Branch, Communicable Disease Center, U.S. Public Health Service, Atlanta 22, Ga.

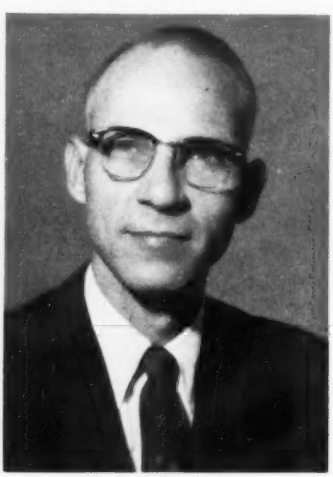
Among the States and Provinces

Arizona

PHOENIX—DR. MACKERY NAMED VETERINARIAN IN CHARGE OF ADE ACTIVITIES.—Dr. E. R. Mackery was appointed veterinarian in charge of the U. S. Department of Agriculture's Animal Disease Eradication activities in Phoenix, Ariz., in August 1959.

Dr. Mackery was formerly stationed at San Juan, Puerto Rico, as veterinarian in charge of the Division's animal inspection and quarantine department and meat inspection activities—a post he had held since July 1, 1956 (*see* the JOURNAL, June 1, 1960, pp. 583-584, for an account of his successor, Dr. L. N. Miller, Jr., Puerto Rico).

A native of Florida, Dr. Mackery received his D.V.M. degree from Alabama Polytechnic Institute (now Auburn University) in June 1942. After graduation, he accepted employment with the former BAI and worked for the meat inspection division on assignments in Oklahoma City, Okla., and Moultrie, Ga.; on the field force in Georgia; and on the United States-Mexico Commission for the Eradication of Foot-and-Mouth Disease in Mexico. He was assistant veterinarian in charge of the ADE Division's field station, in Atlanta, Ga., prior to his transfer to Puerto Rico in 1956.

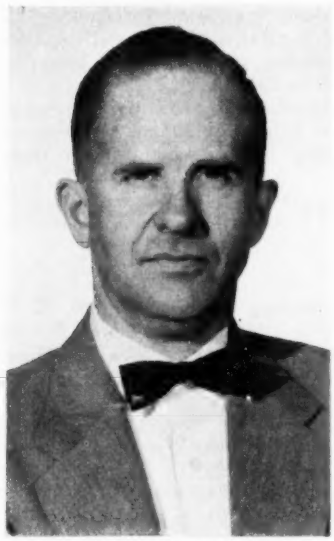


Dr. E. R. Mackery

Dr. Mackery succeeds Dr. Donald Miller who was transferred to the Washington, D. C., office.

Colorado

DR. J. R. COLLIER SUCCEEDS DR. A. W. DEEM IN C.S.U.'s VETERINARY COLLEGE.—Dr. John R. "Ray" Collier, a 1941 graduate of Ohio State University, has been named



Dr. John R. Collier

head of the Department of Pathology and Bacteriology at the College of Veterinary Medicine, Colorado State University, according to an announcement by Dean Rue Jensen. His appointment was effective July 1, 1960. He succeeds retiring Dr. Arthur W. Deem (CIN '18).

Dr. Collier grew up on a farm near Marysville, Ohio. He entered the Armed Forces in 1941, serving mostly at Carlisle Barracks, Pa., where he was a staff member of the medical field services school.

He left the Army in 1946 and began a large animal practice in Minnesota. Two years later, he entered Iowa State College where he was employed in the Iowa veterinary diagnostic laboratory and in the Department of Veterinary Hygiene. He received a Master of Science degree from that institution in 1951 and a Ph.D. degree in 1955.

Dr. Collier has been engaged principally in teaching and research work in animal disease at Colorado State University since his arrival in 1956.

He is a member of the AVMA, the Society of American Bacteriologists, the United States Livestock Sanitary Association, Sigma Xi, Pi Kappa Phi, and Phi Zeta.

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FORT COLLINS—AEC AWARDS RADIATION GRANT TO COLORADO STATE UNIVERSITY.—A grant from the Atomic Energy Commission has recently been made to the Agricultural Experiment Station at Colorado State University for the study of radiation effects on the semen quality of bulls.

The work will be conducted by Dr. W. D. Carlson, associate professor of radiology in the College of Veterinary Medicine, and by Dr. Frank Gassner, professor of chemistry in the endocrine section of the Experiment Station there.

Florida

JACKSONVILLE—DR. J. B. HEALY APPOINTED VETERINARIAN IN CHARGE.—The appointment of Dr. John B. Healy as veterinarian in charge, Animal Disease Eradication Division, ARS, USDA, became effective April 4, 1960. Dr. Healy was formerly veterinarian in charge, ADE Division in Frankfort, Ky.

Born in Junction City, Kan., Dr. Healy attended Kansas State University and obtained his D.V.M. degree from there in

Dr. John B. Healy

1944. For three years, he was engaged in private practice in Iowa.

He entered government service in 1947 with a three-year tour of duty in Mexico on the eradication of foot-and-mouth disease. He held assignments with the Division in Arkansas, Beltsville, Md., and Ohio. He served as assistant veterinarian in charge for four years in Virginia. In June, 1956, he became veterinarian in charge, in Frankfort, Ky., and served in that capacity until his transfer to Jacksonville.

Illinois

URBANA—EASTERN ILLINOIS ASSOCIATION HOLDS SPRING CLINIC.—The "Spring Clinic" of the Eastern Illinois V.M.A. was held on May 13, 1960. All sessions were held in the large animal clinic, College of Veterinary Medicine, at the University of Illinois.

Participating in the program were: Drs. B. W. Kingrey and P. T. Pearson, both of Iowa State University, Ames; James Smith, Waverly; W. G. Raudabaugh, Piper City; L. E. Gambrel, Winnebago; R. K. Shideler, Danville; A. O. Griffiths, Urbana; and H. A. Reynolds, Urbana.

Association members, Drs. Erwin Small, James Meyer, and Harold Heffernan, prepared an excellent one-day clinic for which 72 persons registered.

s/R. D. HATCH, *Secretary.*

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CHAMPAIGN—REPORT OF THE EASTERN ASSOCIATION'S JUNE MEETING.—The Eastern Illinois V.M.A. met at the Hotel Tilden-Hall on June 2, 1960. Members and guests attending numbered 54.

Dr. D. E. Becker of the Department of Animal Science, University of Illinois, discussed "The Hog Producer of the Future." Dr. R. K. Shideler who had so ably represented the Association on the executive board of the state association announced his resignation as he is leaving the area. Dr. E. E. Lutz of Champaign was elected to fill the unexpired term of office vacated by Dr. Shideler.

s/R. D. HATCH, *Secretary.*

Indiana

INDIANAPOLIS—CENTRAL ASSOCIATION PLANS SEMINAR IN FALL.—The Central Indiana V.M.A. will hold its annual small animal seminar on Sept. 14, 1960, at the Claypool Hotel. This will include an afternoon and an evening program with a banquet between the two sessions.

In addition, there will be a program for the women who attend with their husbands.

s/P. T. PARKER, *Chairman.*

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LAFAYETTE—PURDUE TO HOLD DEDICATION CEREMONIES AND CONFERENCE IN OCTORER.—The School of Veterinary Science and Medicine at Purdue University will sponsor the 48th annual conference for veterinarians, Oct. 12-14, 1960.

On October 12, dedication ceremonies of the new School's buildings will be held and a banquet is also scheduled for that evening.

s/ERSKINE V. MORSE, *Dean, School of Veterinary Science and Medicine.*

Iowa

MT. PLEASANT.—On May 5, 1960, the Southeast Iowa V.M.A. held a dinner in honor of five veterinarians of the area who have been engaged in the practice of veterinary medicine for 50 years.

Honored were: Drs. Charles W. Wiley (KCV '10), Farson; Jay M. Wilson (CVC '09), Winfield; John B. Gingrey (KSU '10), Muscatine; H. M. Griffin (CVC '09), Morning Sun; and Clifton C. Logan (ISU '12), Ottumwa.

On June 5, 200 friends and neighbors of Dr. and Mrs. Wiley paid tribute to them at a public reception. In the fall, the Wileys will also observe their 50th wedding anniversary.

s/ GRANT B. MUNGER, *Correspondent.*

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CEDAR RAPIDS—EASTERN IOWA V.M.A. MEETS.—Approximately 300 veterinarians and interested individuals attended the 26th annual clinic of the Eastern Iowa V.M.A., May 10, 1960, at Hawkeye Downs.

The Association's program consisted of the following speakers: 1) diagnostic section—Drs. Charles Pfaff, Cedar Rapids; H. C. Smith, Sioux Falls; J. D. Ray, White Hall, Ill.; W. H. Beckenhauer, Lincoln, Neb.; Ted Bartley, Cedar Rapids; John Dick, Fort Dodge; 2) poultry section—Richard Baum, Osage; W. C. Hillman, Perry; J. R. Ibsen, Charles City; J. P. Jorgenson, Cedar Falls; 3) small animal section—Merrill Vanderloo, Dubuque; Robert Glotfelty, Cedar Rapids;

R. J. Cowles, Burlington; 4) equine section —Oliver Whitcomb and David Bromwell, both of Center Point; 5) sheep section—Dale Brinkmeyer, New London; W. L. Andrews, Milton; Jess Irwin, Iowa City; 6) bovine section—Harold Osbourne, Gladbrook; Paul Ehrig, Reinbeck; G. W. Rieke, Victor; W. R. Goodwin, Newhall; Alex Hoag, Coin—internal fixation of the vagina; Gordon J. Iverson, Harvard, Ill.—tail breeding; Richard Lundvall, Ames—carcinoma of the eye; 7) swine section—Bert Combs, Conrad —hysterectomy for repopulation program; James Pirie, Cedar Rapids—artificial insemination; Maynard Spear, Ames—production testing and certification.

Dr. W. L. Stroup of Corinth, Miss., came the farthest of all the speakers; his topic concerned restraint and gadgets.

s/Grant B. Munger, *Correspondent.*

Kansas

Manhattan—The late Dr. W. M. McLeod's Portrait Presented to K.S.U.—A portrait of the late Dr. William Max McLeod was presented to the Kansas State University School of Veterinary Medicine, Dr. E. E. Leasure, dean of the School, announced, June 10, 1960. Presentation of the portrait was made under the auspices of a McLeod Memorial committee.

At the time of Dr. McLeod's death in 1957, he was head of the department of Anatomy. Prior to his death, he had been a member of the staff at K.S.U.'s School of Veterinary Medicine for 38 years.

Kentucky

Frankfort—Dr. L. T. Fisher Promoted to Veterinarian in Charge.—The appointment of Dr. LeRoy T. Fisher as veterinarian in charge, Animal Disease Eradication Division, Frankfort, Kentucky, became effective on April 3, 1960. He was formerly assistant veterinarian in charge at Little Rock, Ark.

In 1929-1930, Dr. Fisher attended Tulsa University, studying engineering. He entered Oklahoma State University (then Oklahoma A. & M.) in 1931 and received a B.S. degree in biological science. He then enrolled in Ohio State University and received his D.V.M. degree in 1937.

Dr. Fisher entered federal service as a veterinary livestock inspector in the tuberculosis and brucellosis programs in New Jersey. He has worked at the New Jersey, Florida, Mississippi, Louisiana, and Arkansas stations. In addition to tuberculosis and brucellosis programs, assignments have included AIQ work, tick eradication, and vesicular exanthema. Upon completion of the Second Veterinary Administrator Development Program, he was assigned as assistant in charge of field activities in Arkansas.

Dr. LeRoy T. Fisher

Missouri

St. Louis—Greater St. Louis Association Elects Officers.—At the annual meeting of the Greater St. Louis V.M.A. on June 3, 1960, the following slate of officers was elected: Drs. L. N. Atkinson, president; E. E. Epstein, vice-president; H. C. Eschenroeder, secretary; and J. Seelbach, treasurer.

s/H. C. Eschenroeder, *Secretary.*

North Carolina

Raleigh—Eastern Association Elects New Roster.—At the regularly scheduled meeting of the Eastern North Carolina V.M.A. on April 26, 1960, at the Charcoal Steakhouse, the following officers were elected: Drs. C. B. Randall, Kinston, president; Robert Glass, Jacksonville, vice-

president; and Byron H. Brow, Goldsboro, was re-elected secretary-treasurer.

s/Byron H. Brow, *Secretary.*

Pennsylvania

Philadelphia—Fifth Animal Disease Conference for Regulatory Veterinarians. —Veterinarians from the Middle Atlantic states who are responsible for regulating and supervising governmental programs to reduce animal diseases transmissible to man met on May 27, 1960, at the School of Veterinary Medicine University of Pennsylvania. The all-day program dealt with "The Responsibility of the Regulatory Veterinarian in Human and Animal Health."

More than 100 federal and state regulatory veterinarians attended. Program participants and their respective subjects included: Drs. C. G. Durbin, veterinary medical director, Food & Drug Administration—antibiotics and pesticides in milk and animal tissues; Ernest E. Saulmon, ADE Division, USDA—anaplasmosis—its control and eradication; Albert F. Ranney, ARS, USDA—epizootiology with regard to tuberculosis

Some of the program participants are shown relaxing between sessions at the fifth annual animal disease conference for regulatory veterinarians sponsored by the University of Pennsylvania School of Veterinary Medicine.

(Left to Right) Dr. Mark W. Allam, dean, School of Veterinary Medicine; Dr. Samuel F. Scheidy, AVMA president; Dr. Edwin D. Tuckerman, district veterinarian, Pennsylvania Bureau of Animal Industry.

Saskatchewan

Star Phoenix Photo

Executive members of the Saskatchewan V.M.A. for 1960.

Standing (left to right)—Drs. E. E. Carlson, V. E. Senior, R. B. Gaskin, Robert Connell, E. S. Starrak.

Front row—Drs. W. Turnbull, secretary-treasurer and registrar; Harold Struthers, president; J. C. McIsaac, vice-president; T. V. Johnson and G. F. Hamilton.

Dr. H. J. Hunter is missing from the picture.

eradication; Robert R. Marshak, University of Pennsylvania—demonstration of tuberculin reactions; Edwin L. Brower, N.J. Department of Agriculture—regulatory program for eastern encephalitis in New Jersey; Samuel F. Scheidy, AVMA president—AVMA and regulatory veterinary medicine; James L. Hourrigan, special diseases, ADE Division, USDA—scabies, control with special reference to cattle; Francis J. Mulhern, ARS, USDA—report of hog cholera eradication on a national basis; and James F. Murphy, University of Pennsylvania—controlling mastitis.

Virginia

BLACKSBURG—SOUTHWEST ASSOCIATION MEETS.—The Southwest Virginia V.M.A.'s monthly meeting on April 7 featured a film on the new tranquilizer, Librium. Following this, Dr. Blake Fawcett, surgeon at the Radford Community Hospital, spoke on thoracic surgery as done in human beings with application to domestic animals.

A special meeting was held on April 14 in Blacksburg at which time Dr. Mark L. Morris, Allenspark, Colo., discussed nutrition and diet in small animal medicine. Dr. Morris also reported on the activities of the Morris Foundation and the Morris Research Laboratories.

Another regularly scheduled meeting of the Association was held in Blacksburg on May 5, at the Veterinary Science Laboratory. At this meeting, representatives of Valleydale Packers spoke to the group on the integrated hog operation which they are setting up in this area of Virginia.

Drs. E. A. Cahill and Jack Russell from Allied Laboratories, Pitman-Moore Division, also addressed the group. Discussion centered on the ways and means of supplying veterinary services to these operations.

s/D. F. WATSON, *Head, Veterinary Science, Virginia Polytechnic Institute*

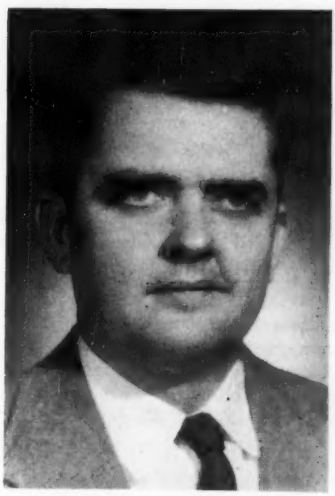
Foreign News

Mexico

Mexico City—DR. D. L. WILLIAMS TRANSFERRED FROM SEBRING, FLA.—Dr. D. L. Williams (TEX '45) was transferred from Sebring, Fla., to Mexico as co-director of the Mexican-American commission for the prevention of aftosa, effective Aug. 9, 1959. Dr. Williams was formerly assistant veterinarian in charge in Florida working exclusively on the joint state-federal screwworm eradication program.

From 1948 to 1952, he served in Mexico as area supervisor and assistant district supervisor. In 1952 Dr. Williams entered Tulane University School of Graduate Medicine, receiving his Master of Public Health degree in 1953. From 1953 to 1955, he worked in Mexico as a field veterinarian and later became district supervisor during a foot-and-mouth disease epizootic in Veracruz.

Dr. Williams has had special training by the Animal Disease Eradication Division in handling epizootics of mucosal disease, bovine rhinotracheitis, and radiological defense training.



Dr. D. L. Williams

Commencements

Graduating Class, 1960, College of Veterinary Medicine, Michigan State University

Michigan State University.—At the 1960 commencement exercises of the College of Veterinary Medicine, Michigan State University, the following 59 candidates were presented for the D.V.M. degree:

Vilas L. Allen
William H. Armstrong
Carl F. Bach
Hermann Bonasch
Quentin W. Bosworth
James W. Buchanan
Raymond R. Burrowes
Arthur B. Butterfield
Charles F. Coussens
Robert M. Diener
John J. Drives
Henry C. Fallis
Ronald R. Flansburg
Lloyd S. Goyings
Ervin Green
John T. Grzyb, Jr.
George R. Gunderson
Ronald J. Haeger
Beverly Ann Holt
John D. Hottell
David G. Howell
James L. Jones
Andrew S. Kalmus
Max R. Kennedy
Lloyd Konyha
Robert M. Kovatch
Joseph J. Kowalski
John W. Kramer
Larry W. Laudig
Peter Lederer
Max A. Lehman
Charles G. Liddle
Darold F. McCalla
Bruce M. Madren
James L. Mahan
Robert L. Mahr
Lowell P. Malmquist
Thomas A. Miller
James Miyat
Leonard J. Perry
Alan R. Ramsey
Donald C. Randall
James A. Reaume
John F. Rigg
Ricardo Sasso
Theodore F. Schaub
William M. Schreer
Ross D. Scoggins
Charles O. Smith
Kenneth E. Smith
Robert E. Spalding
Howard D. Stowe
Raymond E. Sytek
John T. Thornton
Ethard W. Van Stee
Russell W. Wagner
Donald D. Wathen
Robert D. Williams
Richard L. Witter

Graduating Class, 1960, College of Veterinary Medicine, University of Minnesota

Top row (left to right)—Arnold D. Alstad, Steven W. Anderson, Raymond P. Axtman, Ervin J. Baas, Ivan E. Berg, Gerald N. Beste, Charles E. Blackbourn, Robert T. Boschert, Jerry E. Butts.
Second row—Bennie O. Carlson, Sigmund J. Cysewski, Jr., Donald M. Dachel, David M. Duffin.
Third row—Arnold E. Follstad, Barbara J. Follstad, Roger D. Fortney, Perry Gehring, Francine Gough, Bruce W. Gutzmann.
Fourth row—Bejamin L. Hart, Lee A. Hildman, Paul D. Holmberg, Kenneth H. John, Dr. W.T.S. Thorp, dean, LaRue W. Johnson, John K. King, Eugene L. Kirshbaum
Fifth row—Vaughn L. Larson, Warren H. Luedtke, Donald W. Maas, Kenneth G. Magnuson, Joseph R. McGlynn, Arthur Moats, Jr., DuWayne E. Molnau, Harley W. Moon, Robert A. Nelson.
Sixth row—David P. Olson, Ned E. Olson, Richard S. Olson, Donald S. Opstad, Gary R. Sampson, William F. Schwarze, Robert J. Sigfrid, Rolland R. Tibbits, Robert A. Williams.

University of Minnesota.—At the 1960 commencement exercises of the College of Veterinary Medicine, University of Minnesota, the following 44 candidates were presented for the D.V.M. degree:

Arnold D. Alstad
Steven W. Anderson
Raymond D. Axtman
Ervin J. Baas
Ivan E. Berg
Gerald N. Beste
Charles E. Blackbourn
Robert T. Boschert
Jerry E. Butts
Bennie O. Carlson
Sigmund J. Cysewski, Jr.
Donald M. Dachel
David M. Duffin
Arnold E. Follstad
Roger D. Fortney
Perry J. Gehring
Eva F. Gough
Bruce W. Gutzmann
Benjamin L. Hart
Lee A. Hildman
Paul D. Holmberg
Kenneth H. Johnson
LaRue W. Johnson
John K. King
Eugene L. Kirshbaum
Vaughn L. Larson
Warren H. Luedtke
Donald W. Maas
Kenneth G. Magnuson
Joseph R. McGlynn
Arthur E. Moats, Jr.
DuWayne E. Molnau
Harley W. Moon
Robert A. Nelson
David P. Olson
Ned E. Olson
Richard S. Olson
Donald S. Opstad
Barbara J. Persson
Gary R. Sampson
William F. Schwarze
Robert J. Sigfrid
Roland R. Tibbits
Robert A. Williams

Graduating Class, 1960, School of Veterinary Medicine of the Province of Quebec, University of Montreal

Outside border, reading counterclockwise—R. Lavoie, J.-E. Bélisle, C. Hébert, L. Piché, J.-P. Dufault, B. Mital, R. Vrancken, J.-L. Briand, R. Giordano, I. Platonow, C. Berthélémé, J.-L. Flipot, J. Godu, D. Todorov, M. Côté, V. Fincati, M. Délucé, J. Leblanc, S. Cloutier, G. Fritsch.

Center, top two rows, reading the top line first—Dr. J. Saint-Georges, Dr. J. D. Nadeau, Mgr. L. Lussier, Dr. L. Cournoyer, Dr. E. Jacques, new director of the School, and the late Dr. G. Labelle (see the JOURNAL, May 1, 1960, p. 474).

Center, bottom two rows, reading the top line first—A. Brassard, R. Tardif, R. Cournoyer, G. Farly, L. G. Bernier, E. G. Clark, P. Brisson, and A. Marchessault.

University of Montreal.—At the 1960 commencement exercises of the School of Veterinary Medicine of the Province of Quebec, the following 28 candidates were presented for the D.V.M. degree:

J.-E. Bélisie
Gilles Bernier
Charles Berthélémé
J.-André Brassard
Jean-Louis Briand
Pierre Brisson
Erle G. Clark
Sylvio Cloutier
Michel-G. Cote
Rober N. Cournoyer
Marc Délucé
Jean-Paul Dufault
Guy S. Farly
V. Fincati
Jean-Louis Flipot
Gérard Fritsch
Robert Giordano
Claude Hébert
René Lavoie
Jude Leblanc
André Marchessault
Louis O. Piché
Igor Platonow
Rémi Tardif
Dimo A. Todorov
Eric R. Vrancken

• • •

Oklahoma State Unversity.—At the 1960 commencement exercises of the College of Veterinary Medicine, Oklahoma State University, the following 37 candidates were presented for the D.V.M. degree:

Gerald L. Appelgate
James E. Boyd
Cyril M. Brown
Rodye E. Butler
William F. Carter
James A. Countryman
Ralph E. Doutey
James J. Eischen
John J. Fletcher
Kent J. Fletcher
John M. Harper, Jr.
David K. Haviland
Charles L. Heaton
L. A. Holley
John H. James
Michael Kahanovsky
Kenneth G. Keenum
William E. Knighten
Marvin R. Leighton
Larry D. Major
Henry W. Mappes
Harold V. Miller
Culver D. Moe
Don R. Morrow
Thomas M. Mowdy
William D. Munson
Ben B. Norman
Kenneth B. Redmond
William A. Ridenour
Milton C. Schulze
Frank A. Serra
James A. Shmidl
Ernest L. Stair, Jr.
William C. Terry
Billy H. White
Raymond G. White
Howard L. Whitmore

Graduating Class, 1960, College of Veterinary Medicine, Oklahoma State University

Top row (left to right)—Larry D. Major, Michael Canaan, William E. Knighten, Marvin Leighton, David K. Haviland, James Countryman, James Shmidl.

Second row—William C. Terry, John Fletcher, James Eischen, Harold Vardaman Miller, Kenneth B. Redmond, Charles L. Heaton, Ernest L. Stair, Jr.

Third row—William D. Munson and Culver Moe.

Fourth row—Harry James, Ralph Doutey, Henry W. Mappes, Howard L. Whitmore, Lavelle Holley, William Ridenour, Frank A. Serra.

Fifth row—Milton C. Schulze, Sr., Rodye Butler, R. Gene White, Cyrill Brown, Kenneth Keenum, William Carter, Don R. Morrow.

Sixth row—Ben B. Norman, Tom Mowdy, Gerald L. Appelgate, Billy Hunt White, John M. Harper, Jr., James Boyd, Kent J. Fletcher.

Graduating Class, 1960, School of Veterinary Medicine, Tuskegee Institute, Alabama

Top row (left to right)—Bobby D. Barber, Charles A. Blackwell, Ralph P. Brown, Jerry Clinkscales, James A. Ferguson.
Second row—Alvin G. Foster, Jodie Harvey, T. S. Williams, dean, George A. Hylton, Edward H. Jones.
Third row—Charles E. Kimbrough, Marvin Lynn, Howard L. Mitchell, Booker T. Outland, Reginald G. Parris, Willie A. Pridgen.

Tuskegee Institute.—At the 1960 commencement exercises of the School of Veterinary Medicine, Tuskegee Institute, Alabama, the following 15 candidates were presented for the D.V.M. degree:

Bobby D. Barber
Charles A. Blackwell
Ralph P. Brown
Jerry A. Clinkscales
James A. Ferguson
Alvin G. Foster
Jodie Harvey
George A. Hylton
Edward H. Jones
Charles E. Kimbrough
Marvin Lynn
Howard L. Mitchell
Booker T. Outland
Reginald G. Parris
Willie A. Pridgen

University of Pennsylvania.—At the 1960 commencement exercises of the School of Veterinary Medicine, University of Pennsylvania, the following 40 candidates were presented for the D.V.M. degree:

Lester L. Beck
Arthur A. Bickford
Payson J. Brett, Jr.
Ralph L. Brinster
James R. Buell
Robert C. Colladay
Pierre A. Conti
John E. Del Favero
Marylin R. Ernst
George H. Eyrich
Carol H. Fegley
Donald D. Ginand
Orland D. Good, Jr.
Elaine P. Hammel

Leonard Haus
Paul M. Herr
James H. Hughes
Richard W. Kennedy
Harvey A. Kryder, Jr.
William E. Kwaak
Robert L. Lash
Bryan Lee
Julius Melbin
Edward Mikus
Jordan M. Miller
Russell E. Pierson
Frederick J. Price
William J. Prothero
Carl C. Reynolds
Clarence R. Rothrock
Larry A. Schmuck
Douglas N. Sorenson
Clyde S. Streett
William A. Suro
Charles J. Swider, Jr.
Eugene E. Toothaker
Stuart E. Wiles
Walter M. Woolf
Ralph L. Yergey
Jack W. Zimmerly

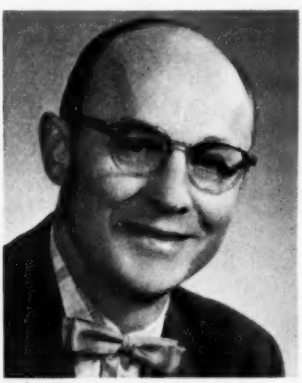
(Pennsylvania did not submit a picture of its graduating class)

Deaths

Star indicates member of AVMA

★ **Russell J. Beamer** (ISC '40), 47, died May 25, 1960, in Houston, Texas.

Dr. Beamer, professor and director of the Small Animal Clinic in the School of Veterinary Medicine, A. & M. College of Texas since 1958, was born in Bennett, Iowa, and attended Iowa State College. After 14 years of general and small animal practice in Iowa and Ohio, he became a lecturer at the A. & M. College of Texas in 1954. He was awarded his master's degree in 1958.



Dr. Russell J. Beamer

Dr. Beamer was affiliated with the Brazos Valley V.M.A., the Texas V.M.A., and the AVMA. He wrote in the areas of surgery, clinical syndromes, and ophthalmology.

★ **Edward Records** (UP '09), 73, died of a heart attack in his laboratory office on the University of Nevada campus, May 13, 1960.

Dr. Records was executive officer of the Nevada State Department of Agriculture and director of the Animal Disease Laboratory. Prior to this appointment, he had been head of the Department of Veterinary Science at the University.

In 1914, Dr. Records was affiliated with the H. K. Mulford Co., Glenolden, Pa., as a bacteriologist when he was asked to come to Nevada to study "red water," a disease which was threatening the cattle industry in Carson Valley.

Dr. Edward Records

What was to have been a six-month project turned into seven years of research and resulted in discovery of the cause and nature of the disease as well as the development of a successful method of immunization which is still in use. Dr. Lyman Vawter was his associate in this work.

Research into many other livestock diseases and the contribution of over 30 publications to the literature of veterinary science helped to fill the 46 years of his service to the state of Nevada. He was an executive officer of the Nevada State Board of Stock Commissioners for many years. He contributed greatly to the animal disease control programs of the state, especially, in late years, in the promotion of the state's campaign for brucellosis eradication.

Dr. Records was a lifetime member of the AVMA and instrumental in the formation of the Nevada State V. M. A. which he served as president for a year and as secretary-treasurer for 12 years.

The Twelfth International Veterinary Congress Prize, the highest honor in the field of veterinary medicine, was awarded to Dr. Records by the AVMA in August, 1957. He was also a member and past president of the Livestock Sanitary Association.

Women's Auxiliary

What to Wear in Denver

August in Denver is probably the warmest month of the summer during the day but the evenings are always cool enough for that pretty sweater or wrap. Some women wear fur stoles in the evening but, unless you are planning to go to Central City for the opera, a summer wrap would be ideal for all other occasions.

Summer clothes and white accessories are still being worn in August but black patent leather shoes or dark summer suedes with dark cottons and fall accessories seem to be more in style. A basic black dress is always suitable.

If you are planning to visit the mountains be sure to include a warm coat or jacket when you pack. The picturesque mountains always look warm and appealing until you step from your car into the cool atmosphere atop the Rockies. It's then that you will wish for "that warm something" you left behind.

Comfortable shoes are also important even if it is only a pair of rubber-soled ones. Pack for comfort and be prepared. See you in Denver!

Ohio

COLUMBUS—O.S.U.'s STUDENT AUXILIARY.—Members of the Women's Auxiliary to the AVMA Student Chapter of the College of Veterinary Medicine at Ohio State University were invited to attend the annual meeting held by the Auxiliary to the state association, Feb. 2, 1960.

Highlights of the meeting for the student wives were the public relations reports and an address by Dr. W. R. Krill, dean, College of Veterinary Medicine at Ohio State.

s/MRS. LOLA OWEN, *Secretary.*

Iowa

AMES.—A tea and election of officers for the Ames Auxiliary to the Iowa V.M.A. were held on April 30, in the veterinary medical laboratories' reading room at Iowa State University.

Officers elected for the 1960-1961 period are as follows: Mrs. R. L. Lundvall, president; Mrs. Matthew J. Eggert, president-elect and delegate to the AVMA Convention in Denver; Mrs. Durwood L. Baker, first vice-president; Mrs. Wayne D. Yoder, recording secretary; Mrs. B. W. Kingrey, treasurer; Mrs. Harry W. Yoder, membership secretary; and Mrs. Ralph W. Mohri, historian.

After the induction of officers, the Auxiliary toured the facilities of the College of Veterinary Medicine at Iowa State.

s/GOLDIE A. (MRS. R.L.) LUNDVALL, *President.*

Newly elected officers of the Ames Auxiliary to the Iowa V.M.A. chosen on April 30 are: left to right—Mrs. Richard L. Lundvall, president; Mrs. Matthew J. Eggert, president-elect and AVMA Convention delegate; Mrs. Durwood L. Baker, first vice-president; Mrs. B.W. Kingrey, treasurer; Mrs. Wayne D. Yoder, recording secretary; Mrs. Harry W. Yoder, membership chairman. Not included in the picture is Mrs. Ralph W. Mohri, historian.

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FURACIN, brand of nitrofurazone (5-nitro-2-furaldehyde semicarbazone), is one of a group of synthetically produced organic compounds known as the nitrofurans—a unique class of antimicrobials. More than 2,500 scientific papers in 25 languages in 40 countries reflect the importance of this group of therapeutic agents in human and veterinary medicine.

The many advantages which have made FURACIN outstandingly effective in an increasing number of clinical indications are:

- bactericidal rather than bacteriostatic—uniquely different from the sulfonamides and most antibiotics
- effective against a wide range of pathogenic bacteria regardless of their gram-staining properties
- little or no development of bacterial resistance
- does not interfere with phagocytosis or healing
- sustained effectiveness in presence of pus, blood or serum

FURACIN is effective in vitro against a multiplicity of organisms including many antibiotic-resistant strains of:

GRAM-NEGATIVE BACTERIA	Proteus sp.*	Clostridium sp.
Aerobacter sp.	Pseudomonas sp.*	Corynebacterium sp.
Alcaligenes sp.	Salmonella sp.	Diplococcus sp.
Brucella sp.	Serratia sp.	Mycobacterium sp.
Escherichia sp.	Shigella sp.	Staphylococcus sp.
Klebsiella sp.		Streptococcus sp.
Paracolobactrum sp.	*GRAM-POSITIVE BACTERIA*	(Sensi-Discs for sensitivity testing are available from Baltimore Biological Laboratory.)
Pasteurella sp.	Bacillus sp.	

*Not all strains of Proteus sp. and Pseudomonas sp. are sensitive in vitro.

The specific action of FURACIN is indicated in necrotic enteritis of swine, gray droppings of mink, avian coccidiosis and bovine mastitis. It is also an effective topical agent against bacterial infections of surface lesions, the eye, ear, and genital tract.

WHAT IS YOUR Diagnosis?

Make your diagnosis from the picture below—then turn the page

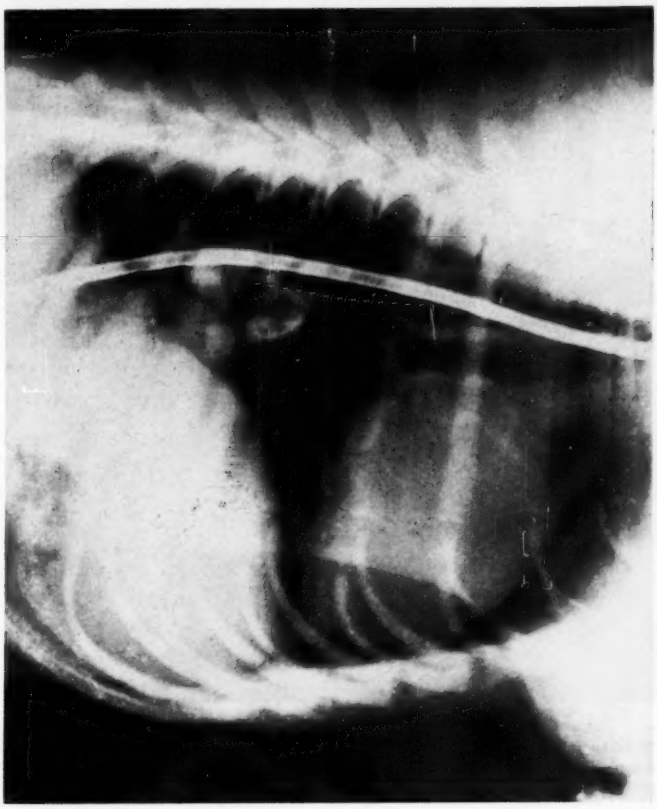

Fig. 1—Lateral recumbent radiograph of the thorax of the dog.

History.—A healthy, male Boxer, 5 months old, developed a cough, then became anorexic and depressed. The body temperature was 104.0 F. and the throat and tonsils were inflamed. Antibiotics were given. In 48 hours the body temperature returned to normal, but the cough became more pronounced and purulent discharge appeared in the nostrils and eyes. The depression continued and the dog did not eat or drink for 2 days. He was coaxed to take small amounts of broth, but after several swallows a small amount of it was regurgitated. Solid food, when forced down, was regurgitated immediately. The dog was anesthetized and a flexible stomach tube, 1 cm. in diameter, was passed down the throat and into the stomach without resistance. A lateral recumbent radiograph of the thorax was taken before the tube was removed (fig. 1).

Here Is the Diagnosis

(*Continued from preceding page*)

Diagnosis.—An irregular foreign object was visible in the thorax in the triangular area ventral to the vertebral column, just anterior to the diaphragm (fig. 2). The stomach tube appeared to be deflected upward and over the object, so it was assumed that the object was lodged in the esophagus just anterior to the hiatus.

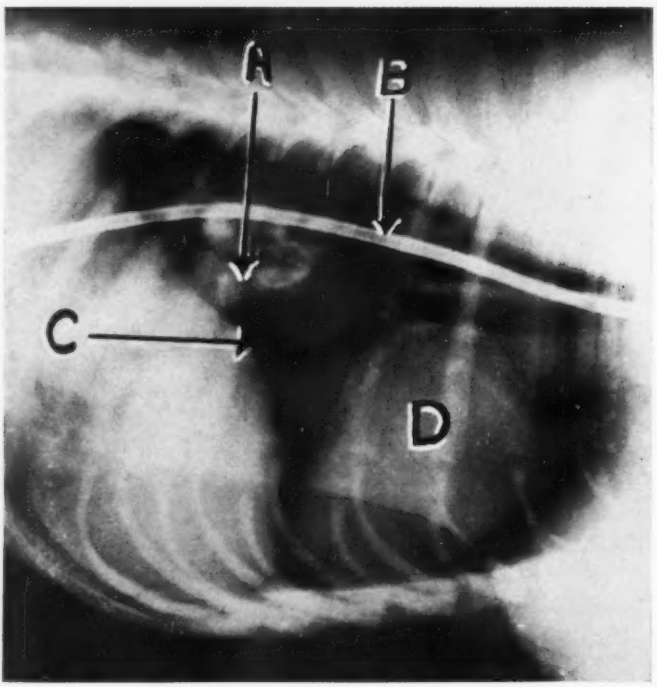

Fig. 2—Lateral recumbent radiograph of the thorax of the dog, showing (A) foreign object which proved to be a bone; (B) stomach tube; (C) diaphragmatic line; and (D) heart.

Comment.—The radiopaque object which proved to be a bone was removed from the esophagus by a forceps through a gastrotomy incision. Recovery was satisfactory.

Bone, metal, and plastic are the chief objects that lodge in the upper digestive tract. Obstruction usually occurs at one of three levels: (1) posterior to the pharynx; (2) anterior to the base of the heart; and (3) anterior to the esophageal hiatus.

The offending object is usually irregular and, because of its shape, only partially blocks the esophageal lumen, allowing liquids and semisolids to pass into the stomach. The mild distress and depression associated with such an obstruction mimics a variety of febrile diseases and adds to the difficulty of obtaining a correct diagnosis. Painful swallowing, excessive salivation, retching, and slight swelling of the area are typical signs of "high choke." Objects in the thoracic area may cause only restlessness, depression, and perhaps fever.

Injury to the esophagus is usually not serious, but sharp objects may cause ulceration of the mucosa or, in rare instances, may pierce the esophageal wall. If perforation does occur, the illness usually terminates in death from septic inflammation of the mediastinum or pleura. Smooth objects usually can be forced into the stomach with a metal gastroscope or can be removed with a forceps, either by way of the pharynx or stomach. In rare instances, thoracic esophagotomy is indicated.

This report was submitted by Drs. J. S. Jasmin, L. H. Jasmin, and J. J. Jasmin, Montreal, Que., and was prepared with the assistance of Wayne H. Riser, D.V.M., M.S., Kensington, Md.

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FURACIN Solution Veterinary—water-miscible liquid of FURACIN 0.2% in polyethylene glycol and water. Valuable in bovine mastitis and impaired fertility.

IN BOVINE MASTITIS FURACIN Solution Veterinary SQUEEJET®—*new*, single-dose, disposable unit of 30 cc. FURACIN 0.2% in polyethylene glycol and water. Especially adapted for use in impaired fertility.

FURACIN-penicillin GEL Veterinary—a crystalline suspension of FURACIN 2% and 13,333 units per cc. of procaine penicillin G in peanut oil with 3% aluminum monostearate.

IN EYE INFECTIONS FURACIN Soluble Powder Veterinary —a soft, water-soluble powder containing FURACIN 0.2% in polyethylene glycol.

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FURACIN Solution Veterinary—water-miscible liquid of 0.2% FURACIN in polyethylene glycol and water.

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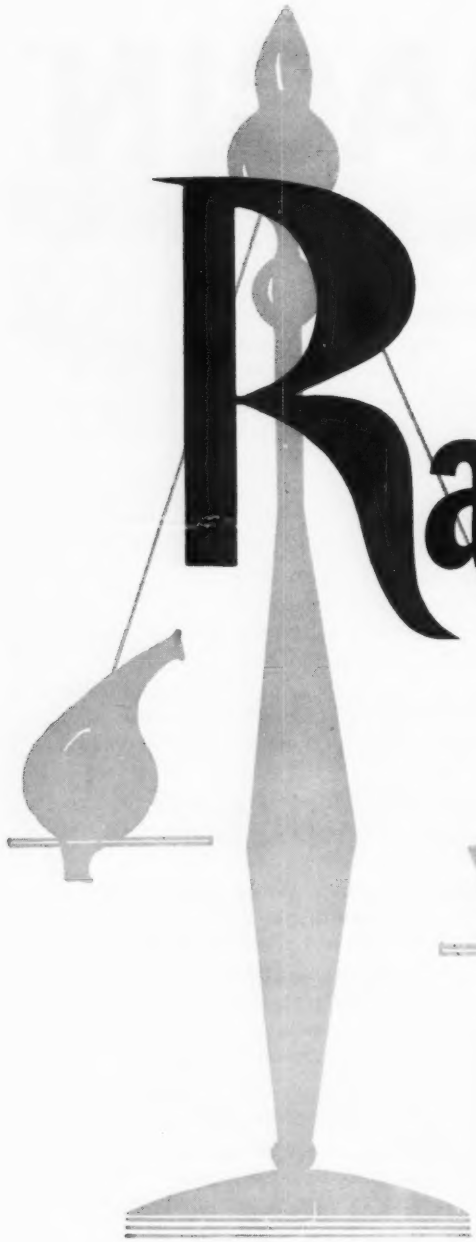

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J. F. Smithcors, D.V.M., Ph.D.

History of the AVMA

1891

The 1891 meeting at Washington, D.C. was somewhat disappointing following the successful Chicago meeting the previous year. Only five "western" veterinarians attended, and just two veterinary colleges and one veterinary science department were represented.

The Special College Committee "made the time-honored report that most of the colleges *favored* a three year college curriculum, but were prevented from attaining their desires through the short-comings of some neighbor." At the time the feeling was (and probably with some justification) that students would desert colleges that announced a three-year curriculum, and: "American veterinarians, with their deficient education and short college courses, yet manage through inherent enterprise and adaptability to maintain a high rank alongside their colleagues from the long-course colleges of other countries."

Papers were presented by C. C. Lyford on "Barren Mares," by W. L. Williams on "Rachitis," and by R. S. Huidekoper on "Identification of Animals."

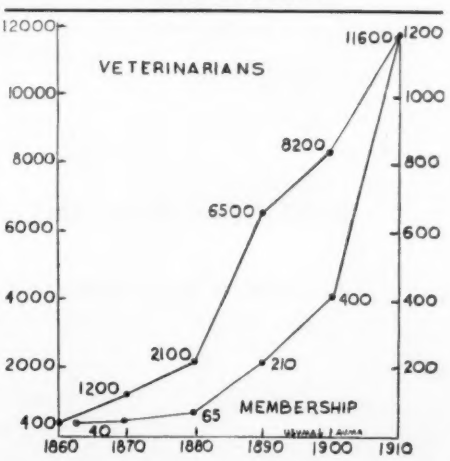
In his presidential address, R. S. Huidekoper observed: "The Association has assumed a size and standing which now make the success of its meetings not only of consequence to the personal comfort and satisfaction of those attending, but the larger meetings and the character of the subjects which are brought before them are reflected over the community, who will judge us and our merits and worth by the opinion which we ourselves form of each other."

A comprehensive, although perhaps not complete, listing of members from 1863 to 1893 gives a total of 550 in these 30 years, of which 302 were listed as active members for 1893. There were 109 listed as dropped for non-payment of dues, 21 deceased, 4 resigned, and 7 expelled. There are obvious discrepancies, however, for only 18 of the 40 founders are listed, and a number of those not listed are known to have died. Seven of the founders were still active members: John Busteed, Charles Burden, Alexandre Liautard, Isaiah Michener, J. Penniman and J. H. Stickney.

The Raynor brothers, Joseph and Thomas, were both present at the organization meeting in 1863, but were among several who withdrew. According to an unpublished manuscript of W. H. Hoskins this was over some question of scruples. Both were known as eminent practitioners of high integrity. In 1864, Robert Jennings attempted to have Thomas Raynor listed as a charter member of the USVMA, but neither he nor his brother became active in Association affairs until the 1880's. For another 20 years or more both were closely identified with veterinary activities in Pennsylvania and with the USVMA, Thomas becoming a vice president a few years before his death in 1908. Their father, who had emigrated from England in 1842, was a veterinarian, as were two other sons; the four brothers each practiced for more than 50 years. James, who died in 1905, is said to have been one of the two or three graduates of the Veterinary College of Philadelphia, headed by Jennings until it closed in 1866.

The officers of the previous year were reelected. The venerable Isaiah Michener, who was honored for his more than fifty years of service to the profession, reminisced: "I attended the first call made for the purpose of forming a veterinary association, which was held in New York in 1863. The idea of forming such a society I think originated in the office of Dr. Jennings in Philadelphia. . . . Several names were suggested, one of which I remember was 'Veterinary Association,' which was vigorously opposed. . . . I then suggested the name of 'United States Veterinary Medical Association,' which was adopted." After noting the progress that had been made, he regretfully observed that "any one who has ever removed the placenta from a cow or found a soft place in her tail to cut into to let the wolf out," could still register as a veterinarian.

Below is a graph showing USVMA membership from 1863 through 1910.



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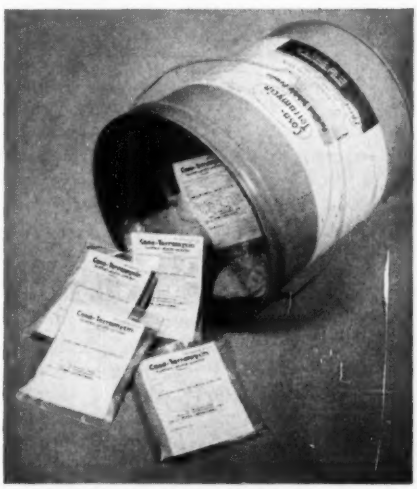

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1,500 mg. pantothenic acid

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Quiz for Quidnuncs

1. How many swine lungs from a herd must be examined to determine freedom from virus pig pneumonia (VPP)? Page 189.

2. What are some of the viral diseases in which inclusion bodies occur? Page 161.

3. What is a maduromycotic mycetoma? What prognosis can be given for this disease in the dog, cat, and horse? Page 201.

4. How is a peritoneopericardial diaphragmatic hernia diagnosed? What surgical technique has proved successful in repairing this type of hernia? Page 181.

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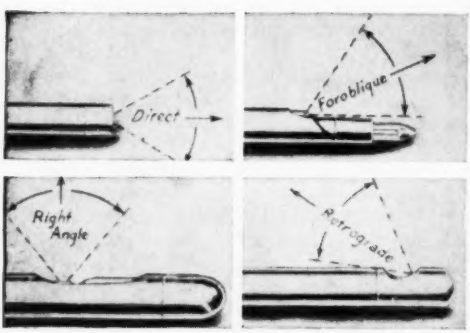
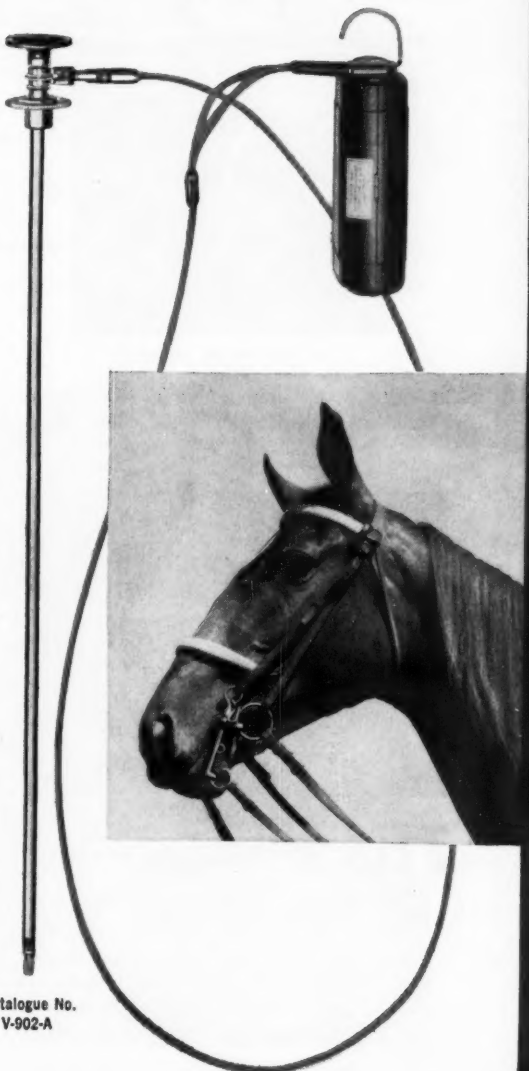
Right angle vision for the examination of the upper respiratory tract of large animals is provided by the specially designed ACMI endoscope shown here. It is .390" in diameter and 50 cm. long. Illumination is supplied by a light conducting cord and a 4-cell battery box. A walnut case protects the instrument when not in use. Endoscopes are also available for the examination of body cavities in small animals.

Other high quality ACMI endoscopes are provided with deflectors to permit catheterization or accommodate forceps for taking biopsy specimens under visualization, and in 4 angles of vision: direct, foroblique, right angle and retrospective. An endoscope having a 6 Fr. diameter can be made in lengths from 3" up to 6"; larger diameters up to 50 Fr. can be made up to 36" or longer.





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Dr. R. W. Bailey Joins Corn States Staff

Robert W. Bailey, D.V.M., has joined the professional staff of Corn States Laboratories, Inc. as a member of the Clinical Evaluation Department. Dr. Bailey, (MSU '43), was stationed at Lackland Air Force Base, Texas, where he was veterinary officer in charge of the Sentry Dog Center. Prior to military service, he was in general veterinary practice in Shawnee, Okla. In 1955 he did postgraduate work in epidemiology at the University of Michigan.

Dr. Robert W. Bailey

Dr. Bailey is a member of the AVMA and Delta Omega.

Lung Cancer in Dogs

In a letter published in the May 7, 1960, issue of the *Journal of the American Medical Association,* Ohio State's Dr. Chauncey D. Leake commented briefly on some conversations he has had with Russian physicians.

Regarding lung cancer, his informants said, in 1956, that there was increased incidence in Russia's large cities but not in rural areas. They felt that cigarette smoking was not alone to blame; their cigarette consumption is much lower than ours and their cigarettes contain less tobacco. Although their cities have smog, as do our own, this is not due to industrial pollution, because all the smokestacks are said to be equipped with precipitators. Although the number of automobiles is relatively small there, it was believed that the numerous buses and trucks were largely responsible for smog.

Last year, Dr. Leake learned from a member of the Institute of Higher Nervous Activity of the Academy of Sciences of the USSR in Moscow that veterinarians had made surveys of the incidence of lung cancer among dogs in large Russian cities and those in rural areas. There was significantly more lung cancer among city dogs than among rural dogs.

Although realizing that the Russians had not supplied specific data and recognizing that they lack statistical sophistication, Dr. Leake felt this information worth considering. Additionally, he commented that such a survey of dogs has probably not been made in this country because members of the medical profession do not consult often enough with those in veterinary medicine.

Dr. Leake has been a friend of our profession for many years and is well aware of the many ways veterinary medicine can contribute to the public health. It would be

gratifying if we could send him, in the near future, a copy of one of our Journals containing an extensive and well-documented survey of the incidence of lung cancer in rural and city dogs.

A report to be published in the September, 1960, issue of the *American Journal of Veterinary Research* adds to the literature 16 more cases of primary pulmonary carcinoma. The source material came from 9,282 necropsies, and the habitat of the dogs was recorded as urban or rural.

Presuming that the necessary data for a more extensive survey might already be recorded at various institutions, several authorities were asked to comment.

One commented that most malignant tumors in the canine lung are metastatic and that a statistically sound study would necessarily involve a large sample of the canine population.

Another respondent pointed out that most primary lung tumors in dogs are adenocarcinomatous, while those in man are of the squamous cell type, possibly a reflection of different causative factors, of which cigarette smoking is only one.

Still another said that one team of investigators had nearly completed such a study, that the data are now being subjected to detailed statistical analysis, and that the study involves not only urban versus rural habitat but will compare data obtained during several decades.

A Quick Getaway • • • —*UPI Photo*

Clyde, at gate 1, and Beatty, at gate 2, thud past the starting gate for the hundred-yard "dash" in the special Jumbo Handicap at Gulfstream Park Racetrack in Hollandale, Fla. Neither contestant caught up with Jungleland who broke fast from gate 3 to win the race.

Public Relations

BRIAN M. FORSTER,
DIRECTOR OF PUBLIC INFORMATION

Speakers' Kits

It Takes More Than Treatment to Control Mastitis, Veterinary Medicine in Our Modern World, Rabies, and *Veterinary-Feed Relations* are titles of existing AVMA speakers' kits. A fifth, titled *The Veterinarian and the Public Health* is a new kit under final review by members of the Council on Public Health and Regulatory Veterinary Medicine.

Three types of AVMA kits are available at no charge to members. The first type deals with specific diseases, the second type covers broad, general areas of the profession, and the third deals with specific areas of public relations.

Speaker's Kit 1

The mastitis kit contains four variations on the theme. One version of the talk is suggested for direction to a general farm group such as a "vo-ag" class, a county agent meeting, or the local grange or farmer's union meeting. The second suggested speech is directed specifically at dairymen who might be gathered for an Aryshire, Brown Swiss, Holstein-Friesion, Guernsey, or Jersey breeders' meeting.

A third talk, directed at a professional audience, is suggested for use at local association meetings. The final variation on the mastitis theme is suggested for use before service clubs or other nonagricultural groups.

In addition to these outlines for talks, the kit contains news releases for use in publicizing the talk before and after presentation. A radio interview script and a television script implemented with pictures and charts are also provided. Pertinent AVMA film library selections on the subject are also listed.

This was the first speaker's kit developed by the AVMA and was introduced in 1955. Since that time, about 75 copies of this kit have been used each year.

Slides are also available to enable a speaker to make a visual presentation. These may also be used on TV if the station is equipped to project them.

Pictured at the left are the five Speakers' Kits, available at no charge to AVMA members.

Speaker's Kit 2

The second speaker's kit is a different type, attempting briefly to expose an adult or a child audience to the many facets and contributions of veterinary medicine to our modern society.

Only two suggested talks are included: one for use before service clubs, PTA's, and similar adult groups, both rural and urban. The second variation is aimed at high school groups for use during career conferences.

The New York State Veterinary Medical Society produced a slide series "The Education and Opportunities of the Veterinarian" which is available with the AVMA kit.

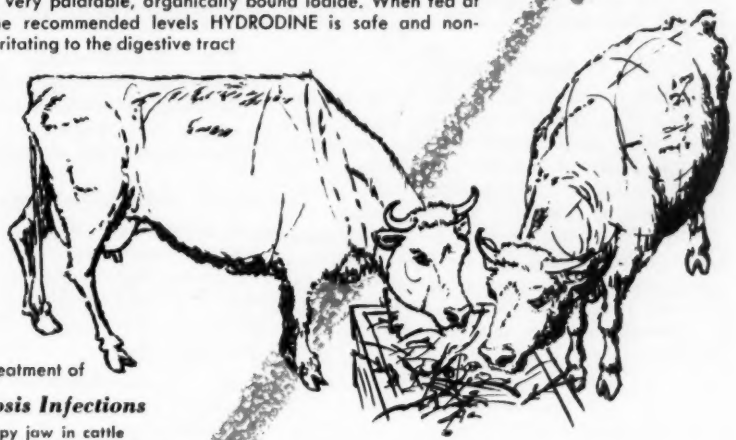
"Veterinary Medicine As A Career," the AVMA booklet, is also included in the kit and additional copies are available through the AVMA headquarters. These may be distributed to the high school students, or the

Continued on adv. p. 42.

dependable!..

HYDRODINE*

a very palatable, organically bound iodide. When fed at the recommended levels HYDRODINE is safe and non-irritating to the digestive tract



A specific for the treatment of

- ***Actinomycosis Infections***
  Lumpy jaw in cattle
  Fistulous withers in horses
- ***Actinobacillosis***
- ***Infectious Pododermatitis***
  (Foot Rot)
- ***Thyroid dysfunction***
- ***Chronic coughing***

also as an aid in the correction of sterility, chronic udder infections and chronic iodine deficiencies. When fed at low levels Hydrodine will help prevent the occurrence of foot rot.

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PR Page—continued from adv. p. 40.

students may be invited to write individually to the AVMA for their individual copy. Those who are sincerely interested will write.

Additional material in the kit includes a chronological history of events in veterinary medicine and brief facts with which to expand the given material, the news releases and the radio and TV material.

Speaker's Kit 3

Recently implemented with charts for use on television, the third kit on rabies includes three talks: one directed to rural audiences, one to urban audiences, and the third to groups concerned with wildlife conservation including the Izaak Walton League.

A seven-minute and a fourteen-minute radio script, catalog of pertinent films, one photo, and charts on rabies are included for TV use together with newspaper releases for announcing the talk and summarizing the material.

Speaker's Kit 4

A departure from the formats of earlier kits, *Veterinary-Feed Relations* provides speech material from talks delivered by officers of the AVMA and the American Veterinary Nutritionists Association before feed manufacturers and joint veterinary-feed meetings.

Additional material includes programs of veterinary-feed meetings. Originally, reprints of articles by AAVN officers appearing in feed magazines, and the results of the AFMA survey conducted among feed manufacturers was included, but this material has run out and is irreplaceable.

Speaker's Kit 5

The newest kit in the speaker's series is titled "The Veterinarian and the Public Health." Talks in the kit are directed to rural, urban, and professional audiences.

A radio and TV script and news releases accompany this kit. Other materials, such as a comic book for child audiences, are available through the Communicable Disease Center, U.S. Public Health Service, Atlanta, Ga.

Film implementation is confined to zoonoses and meat inspection.

A slide series on pathologic conditions found in meat inspection lends itself to use before professional audiences.

Slide Talks Available

A slide library to help speakers appearing before rural and urban non-professional audiences include: the Dog and the Family, the Education and Opportunities of the Veterinarian, Infertility in Cattle, and Swine Repopulation.

Correction

Two publications, *Cornell Veterinarian* and *Avian Diseases,* were unintentionally omitted from the list of national veterinary publications shown on the Public Relations page of the June 1 issue. *Cornell Veterinarian* was established in 1911 and *Avian Diseases* in 1957.

Dr. Iain Paton Named Director of Professional Services at Jen-Sal

Dr. Iain Paton (GLA '58) has been appointed director of professional services for Jensen-Salsbery Laboratories, Inc., Kansas City, Mo.

In his new position, Dr. Paton will edit Jen-Sal's two veterinary publications and will serve as chief veterinarian in providing technical counsel to the marketing department.

For the past 1½ years, Dr. Paton has worked in the Research Department at Jensen-Salsbery in the field of experimental medicine.

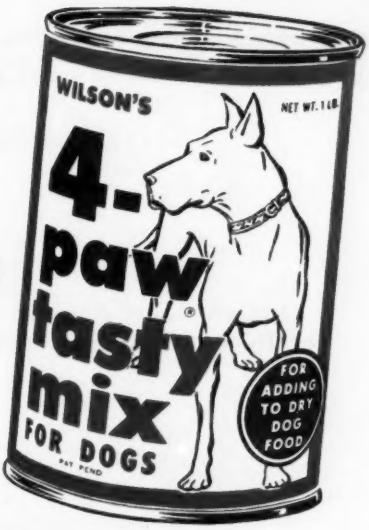
Dr. Paton conducted a practice in Ayrshire, Scotland, for ten years before coming to the United States.

New meat product for instant mixing with dry dog foods

Now available in 1 lb. cans

(packed 24 to the case)

Still available in 6½ lb. cans
100 lb. pails, or 490 lb. drums



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No cooking—you MIX 4-PAW just as it comes from the can. Completely sterile in the can, 4-PAW needs no refrigeration. After opening, treat like meat. Even the size container is custom-planned for you. Each can is a full 16 ounces, packed 24 to a case. Also available in 6 lb., 8 oz. cans; 100 lb. pails; and 490 lb. drums.

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If you have been feeding horsemeat, you have probably worried about supplies and high costs. 4-PAW is the progressive answer. It will continue to be in good supply at reasonable prices because it is made of meat by-products from government inspected food animals.

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96% of 4-PAW consists of carefully selected meat by-products including generous amounts of liver from our own controlled, meat packing plants. Other ingredients supply calcium, phosphorus and vitamins.

Meat by-products	96%	Phosphoric acid	1.4%
Dicalcium phosphate	2.4%	Red Iron Oxide	0.1%
		Cod Liver Oil	0.05%

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To the right is one of many scenic roads winding through the mountains in Colorado.

Indian Lore Abounds in Rocky Mountain Area

The Rocky Mountain Empire, from the Mexican to the Canadian border, is a scenic wonderland offering rich vacation opportunities for the family. Horseback riding, swimming, boating, fishing, and golf are available to participating sportsmen, women, and children, while Indian reservations and preserves are attractive to followers of Indian lore and American history.

Mesa Verde in southern Colorado provides a study of ancient Indian culture. Living in caves and ledges on the face of a sheer cliff, the now defunct Mesa Verde Indians developed a peaceful, agricultural economy, depending on the inaccessibility of their homes for defense against raiding nomadic tribes.

A Navajo and Hopi reservation includes parts of Colorado, New Mexico, Arizona, and Utah. The Hopi portion of the reserve contains the pueblos for which the southwestern tribes were noted. The Navajo is a nomadic sheepherder living, in some areas, in much the same ways his ancestors did.

North of Colorado, Wyoming offers the spectacular scenery of the Jackson Hole Country and the natural phenomena of Yellowstone National Park. Ordinarily avoided by Indian tribes, the Nez Perces traveled through this taboo land in their effort to shake off Army pursuit in 1877. Led by Chief Joseph from the traditional tribal grounds of the Bitterroot and Snake River country where Idaho, Oregon, and Washington meet, the men, women, and children of Joseph's tribe led the Army a 1,300 mile chase across the Northwest. During their trek, they fought 7 battles and several of these battlefields are marked in Wyoming and Montana.

Colorado, Wyoming, and Montana were the rich hunting grounds of the storied mountain men from 1810 to 1840. Laramie and Sheridan were their company trading posts and then bases for Indian patrols by the Army against the Blackfoot, Cheyenne, and Sioux tribes.

Indian relics, preserved or recreated frontier lore, fishing, and horseback riding in the country's most spectacular scenery await veterinarians and their families en route to or from the AVMA meeting in Denver this August.

Daytime temperatures in the 80's and 90's and nights in the 60's are August forecasts for the area. Weather hot enough to swim, cool enough to sleep without air conditioning, makes this region a favorite with many vacationers.

In the distance is Pikes Peak as seen from the Garden of the Gods in Colorado Springs, Colo.

OUTSTANDING!

wherever you find veterinarians!

See you at the AVMA National Convention!

Research Laboratories, Inc., Saint Joseph, Missouri

When you are selecting an antibiotic or steroid for use in treating eye disorders, may we suggest the following Alcon Sterile Ophthalmic preparations, and some points concerning their use.

Three Steroids

ISOPTO® CORTISONE, 0.5% and 2.5%, 5cc Drop-Tainer®.
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ISOPTO® STEROFRIN, 0.5% hydrocortisone and phenylephrine, 5cc Drop-Tainer®.

The first two items are indicated in inflammatory lesions of the anterior segment of the eye (corneal ulcers, iritis, uveitis), allergic conjunctivitis and hypopyon. ISOPTO® STEROFRIN is indicated in ocular allergies (such as might be encountered after exposure to wind and dust), and other inflammatory conditions in the absence of infection.

Steroids are *contraindicated* in the presence of Herpes Simplex. *Caution* should be exercised in the use of ophthalmic preparations containing steroids, because they inhibit the inflammatory response of ocular tissues, and therefore tend to mask an infection.

An Antibiotic-Steroid Combination

ISOPTO® P-H-N (polymyxin, 0.5% and 1.5% hydrocortisone, neomycin), 5cc Drop-Tainer®.

Indicated in acute catarrhal, allergic, bacterial and traumatic conjunctivitis; meibomianitis; blepharitis; keratitis; herpes zoster ophthalmicus; phlyctenular keratoconjunctivitis; mild acute and chronic iritis; and, recurrent marginal ulceration.

An Antibiotic

ISOPTO® P-N-P (polymyxin, neomycin and phenylephrine), 5cc Drop-Tainer®.

The same formula as ISOPTO® P-H-N, *without* hydrocortisone, and using phenylephrine, which is anti-inflammatory without masking infection. Indicated in wounds of the eyelids, follicular conjunctivitis, ulceration of the cornea, external bacterial infection of the eye, and when steroids are contraindicated.

The five items mentioned above, and the complete Alcon line, may be promptly obtained from:

ALBANY SERUM COMPANY, Albany, Georgia
BARBER VETERINARY SUPPLY, Richmond, Virginia
CALIFORNIA MEDICAL SUPPLY, Pasadena, California
CENTRAL CITY CHEMICAL, San Francisco, California
FLORIDA VETERINARY SUPPLY, Tampa, Florida
MILLER VETERINARY SUPPLY, Fort Worth, Texas
NORTHWEST VETERINARY SUPPLY, Oregon City, Oregon
PROFESSIONAL VETERINARY SERVICE, Miami, Florida
SHARPE & VEJAR COMPANY, Los Angeles, California
STANDARD VETERINARY PRODUCTS, Palisades Park, N.J.
JACK A. WEBSTER, Wakefield, Massachusetts

You may also order directly from Alcon. For a *professional sample* of any of the above five preparations, write, designating your choice, to Alcon Laboratories, Inc., Veterinary Department, P.O. Box 1959, Fort Worth 1, Texas.

A SPECIAL NOTE: We hope to see you at the annual AVMA meeting in Denver, August 14 - 18. The Alcon booth will be #103.

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AV-7

Academic Rank of Veterinary Faculty Members

A comparison of academic rank of veterinary faculty members for the school years 1951-1952 and 1959-1960 revealed a decided increase in total numbers from 627 to 959, a 34.5 per cent increase. It is interesting that in 1951-1952 the number of assistant professors (146) was greater than the number of associate professors (100); whereas in 1959-1960, their position has reversed, with 186 associate professors and 174 assistant professors.

TABLE 1—Academic Rank of Veterinary Faculty Members (in the United States and Canada)—Comparison of School Years 1951-1952 and 1959-1960

	1951-1952		1959-1960	
	No.	% of total	No.	% of total
Professor	178	28.4	260	27.1
Associate professor	100	15.9	186	19.4
Assistant professor	146	23.3	174	18.1
Instructor	157	25.0	254	26.4
Other*	46	7.3	85	8.9
Total**	627	.....	959	.....

*Includes research assistants, graduate assistants, residents, interns, field men, etc.

**Of the total number of faculty members in 1951-1952, 88 were nonveterinarians; in 1959-1960, 154 were nonveterinarians.

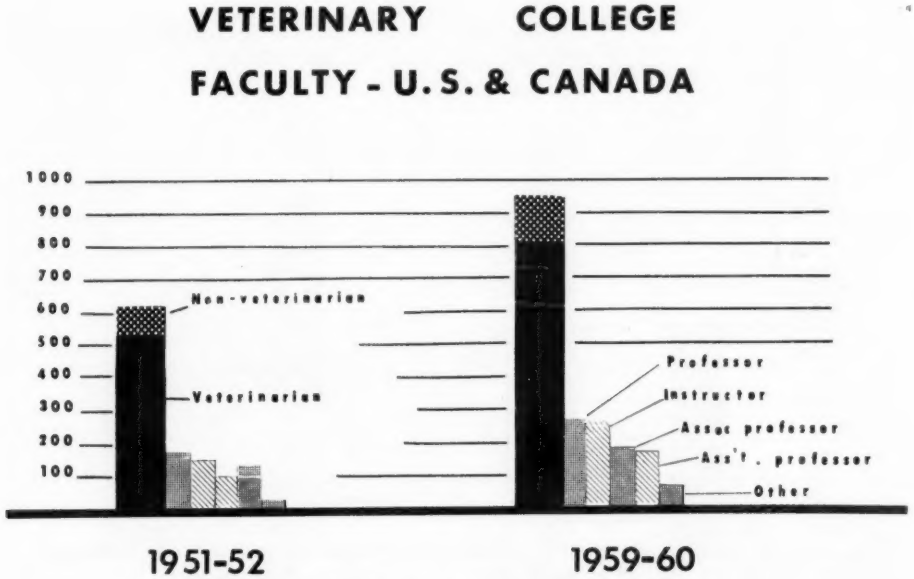


Chart 1—Academic rank of veterinary faculty members.

AVMA Council on Education—1960

Academic Degrees Held by Veterinary Faculty Members

The number of veterinary faculty members with D.V.M. degrees was about 86 per cent of the total faculty in 1951-1952 and about 84 per cent in 1959-1960. The number of faculty members with both M.S. and Ph.D. degrees was not obtainable from the data from which the accompanying table and chart were prepared.

TABLE 1—Academic Degrees Held by Veterinary Faculty Members—Comparison of School Years 1951-1952 and 1959-1960

	1951-1952	1959-1960
Number with D.V.M. degree	539	805
Number with D.V.M. and Ph.D degrees	75	174
Number with D.V.M. and M.S. degrees	161	319
Number without D.V.M. degree	88	154
Number without D.V.M. degree but with Ph.D degree	39	91
Number without D.V.M. degree but with M.S. degree	51	94
Total number with Ph.D degree	114	265
Total number with M.S. degree	212	413

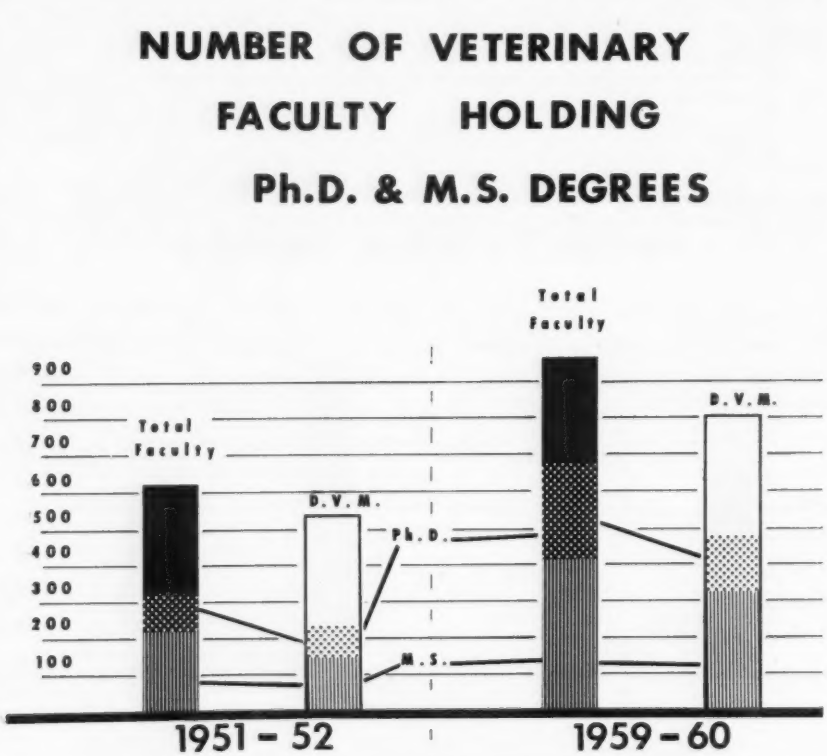


Chart 1—Number of veterinary faculty members with D.V.M., M.S., and Ph.D. degrees.

AVMA Council on Education—1960

AVMA Opposed to Amending Poultry Products Inspection Act

On June 13, 1960, Dr. H. E. Kingman, Jr., AVMA executive secretary, appeared before the Subcommittee on Dairy and Poultry of the Congressional Committee on Agriculture. He stated AVMA opposition to a proposed amendment which would permanently exempt certain poultry products from inspection under the Poultry Products Inspection Act. Dr. Kingman was accompanied by General J. A. McCallam, AVMA Washington Representative. Members of the Subcommittee on Dairy and Poultry present were: Lester R. Johnson, Wisconsin, chairman; Thomas G. Abernethy, Mississippi; Clark W. Thompson, Texas; Harlan Hagen, California; Merwin Coad, Iowa; Robert W. Levering, Ohio; Clifford G. McIntire, Maine; Henry A. Dixon, Utah; Albert H. Quie, Minnesota; Alexander Pirnie, New York.

AVMA concern about the proposed amendment centered not only on exemption of certain poultry products but upon the possibility that this would lead, in turn, to the exemption of other food products from inspection.

Dr. Kingman's statement follows:

The American Veterinary Medical Association does not favor the amending legislation to the Poultry Products Inspection Act. The amendment, if enacted, would establish a permanent exemption to certain poultry products and would remove them from inspection under the Act. This action, we fear, would permit unwholesome and adulterated poultry or poultry products to enter channels of interstate commerce and reach the tables of the consuming public.

On March 7, 1957, the American Veterinary Medical Association, appearing before a similar subcommittee of the 85th Congress, expressed favor and endorsement of legislation which established the Poultry Inspection Act. The statement made at that time appears on pages 97-98 of the report of those hearings.

The Meat Inspection Act and the Poultry Products Inspection Act have provided necessary controls to assure the consuming public a wholesome, clean, unadulterated meat and poultry product when it reaches their tables. These controls have provided for minimum rather than maximum inspection and specify that the inspected product will carry the seal indicating federal inspection. The consuming public has learned to recognize this seal and is confident that the product bearing it meets definite standards of quality. The consuming public is entitled to have controls which adequately protect it when purchasing meat and poultry products. Permitting certain products to be processed without inspection would seriously weaken over-all food inspection program standards.

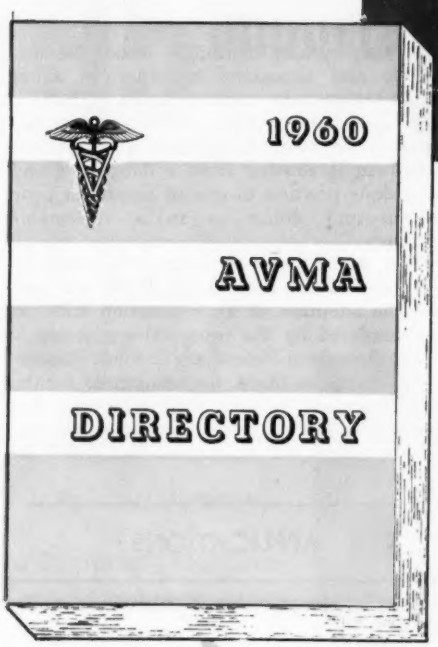
The use of unsafe food additives is called to our attention daily in the public press. Such use of additives constitutes adulteration of food and masks possible unwholesomeness of meat and poultry products. Protection of the public against the use of additives that are unfit for human consumption can be assured only through constant inspection.

Inspection of the facilities in which meat and poultry products are produced is as essential as inspection of the product itself. More than 50 years of experience under the Meat Inspection Act of 1906 has brought about concept of proper facilities for the production of meat products. This concept needs to be constantly reviewed and improved so that the environmental conditions under which these products are produced assures the consuming public of an adequate supply of wholesome, healthful, meat and poultry products. The mark of inspection must be backed up by an inspection program that assures compliance with high sanitation standards.

The exemption provisions included in the Poultry Products Inspection Act which would be affected by enactment of HR 11050 were written into the law to permit on orderly transition from voluntary inspection and uninspected operation of plants to full compliance with the intent and letter of the Act.

It was recognized that, during the period immediately following the enactment of this

(Continued on adv. p. 52)



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Organization Section

Continued from adv. p. 50.

new law, certain flexibility would be desirable and necessary in order to avoid direct violation or noncompliance or both.

No one, we are sure, considered this exemption anything more than a temporary expedient in moving from a dangerous and hazardous position to one of consumer protection—and doing so in a reasonable manner.

The declaration of policy contained in Section 3 of the Act certainly will not permit the adoption of an exemption such as contemplated by the proposed legislation.

The American Veterinary Medical Association wishes to thank the committee for the opportunity to present this statement and urges that the proposed amendment to the Poultry Products Inspection Act NOT be enacted.

APPLICATIONS

Applicants Not Members of Constituent Associations

In accordance with paragraph (c) of Section 1, Article I, of the Bylaws, the names of applicants who are not members of constituent associations shall be published in the JOURNAL. Written comments received by the Executive Secretary from any attive member regarding the application as published, will be furnished to the Judicial Council for its consideration prior to acceptance of the application.

SIMON, K. J.
East Fort
Trichur, Kerala, India
D.V.M., University of Philippines, 1948.
Vouchers: I. A. Merchant and Melvin J. Swenson.

BOWIE, WALTER C.
Box 1127
Tuskegee Institute, Ala.
D.V.M., Kansas State University, 1947.
Vouchers: T. S. Williams and Edward T. Braye.

BLACKLEDGE, GEORGE T.
Box 609
Tuskegee Institute, Ala.
D.V.M., Tuskegee Institute, 1951.
Vouchers: T. S. Williams and Edward T. Braye.

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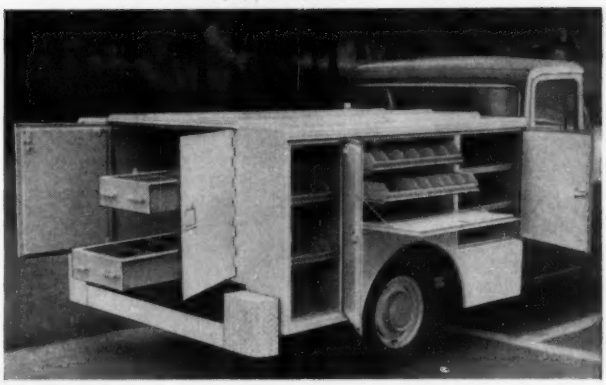

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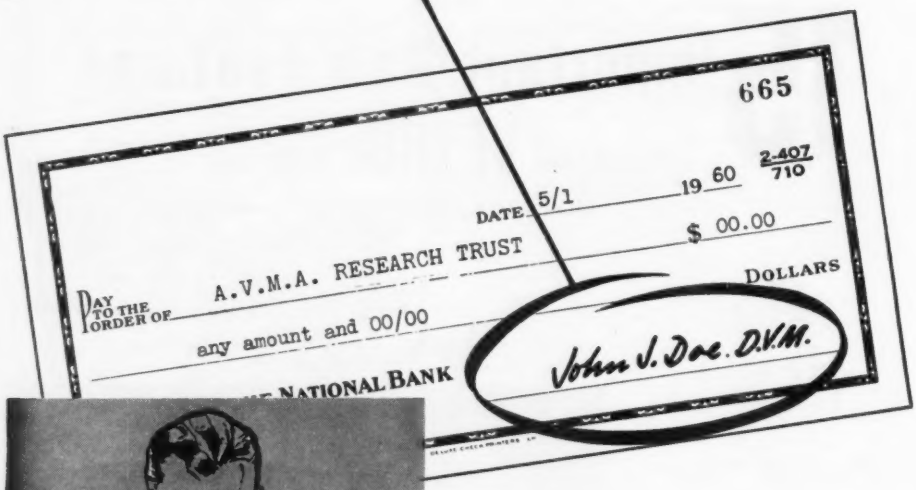

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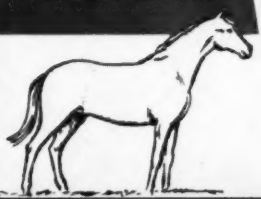

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MAP OF DOWNTOWN DENVER

HOTEL INFORMATION—DENVER, COLORADO, CONVENTION

Ninety-Seventh Annual AVMA Meeting, Aug. 14-18, 1960

All requests for hotel accommodations will be handled by a Housing Bureau in cooperation with the Denver Convention and Visitors Bureau. The Bureau will clear all requests and confirm reservations.

Hotel and Rate Schedule

(See Location, by Number, on Map of Downtown Area)

Map No.	Hotel	Single (1 person)	Double bed (2 persons)	Twin bed (2 persons)	Suites	Sets-2 rooms connecting bath (2-3-4 persons)
1	Adams*	$5.50-7.50	$ 7.50-9.00	$ 8.50-10.50	$13.50-15.00	$ 9.50-16.00 (1 Room, 2dbl. beds)
2	Albany†	6.50-9.50	10.00-12.00	12.50-14.00	30.00	- - - - - - -
3	Ambassador	5.50-6.00	7.00-7.50	9.00	- - - - - - -	- - - - - - -
4	Argonaut*†	6.50-9.50	8.50-11.00	9.50-12.50	- - - - - - -	13.50-17.50
5	Auditorium*	5.00	6.50	7.00	- - - - - - -	8.00-12.00
6	Broadway Plaza†	8.00-10.00	10.00-12.00	12.00-14.50	18.00-28.00	- - - - - - -
7	Brown Palace†	9.00-15.00	13.00-17.00	14.00-19.00	22.00-70.00	18.00-22.00
8	Colorado*	4.50-6.00	6.00-10.00	8.00-12.00	- - - - - - -	14.00-20.00
9	Cory	5.00-7.00	6.00-9.00	6.50-9.00	- - - - - - -	- - - - - - -
10	Cosmopolitan*	8.50-11.00	12.00-18.00	14.00-20.00	22.00-60.00	- - - - - - -
11	Hillview	9.00-11.50	10.00-12.00	12.00-13.50	16.00-18.00	- - - - - - -
12	Hilton*†	HEADQUARTERS HOTEL — Reserved exclusively for official convention use.				
13	Kenmark (not a/c)	4.50-6.50	6.00-7.00	7.50-8.00	- - - - - - -	6.00-12.00
14	Mayflower*	7.50-14.50	8.50-16.50	12.50-18.50	- - - - - - -	- - - - - - -
15	Olin*	5.00-7.00	9.00-11.00	10.00-12.00	- - - - - - -	11.00-16.00
16	Oxford	5.00-10.00	6.50-10.00	8.50-11.00	13.00-16.00	- - - - - - -
17	Sears	5.00-6.00	6.50	7.50	- - - - - - -	14.00 (For 2-3-4 persons)
18	Shirley Savoy*	7.00-9.00	9.50-11.50	11.00-13.00	25.00	15.00-19.00

†100 per cent air-conditioned; in other hotels listed, majority of rooms air-conditioned.

FAMILY PLAN—The above hotels offer a "family plan" whereby children under 12 years of age will be accommodated in the same room with their parents at no extra charge. If more than one room is required to accommodate children, the hotel will charge only the single rate for each room.

MOTELS—Reservations for motels in the Denver area may be made through the Denver Convention and Visitors Bureau, 225 West Colfax, Denver 2, Colo.

PLEASE USE APPLICATION ON REVERSE SIDE FOR HOTEL ACCOMMODATIONS

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The Convention and Visitors Bureau will make every effort to place you according to your expressed wishes or, if the accommodations of your choice are not available, the Housing Bureau will select one that is nearest to the preferred rate and location.

Please give us the complete information requested below. At least four choices of hotels or more if you desire, are necessary. Arrange for double occupancy of rooms wherever possible; only a limited number of single rooms is available.

If you have a few days before or after the convention that you would like to enjoy in the mountains, the Convention and Visitors Bureau will provide you with free information, on request, on the following: sightseeing trip; dude ranches; resorts; housekeeping cabins. In requesting this information, please indicate which type of literature you wish.

ADDRESS: Convention and Visitors Bureau, 225 West Colfax Avenue, Denver 2, Colo.

Reservations will be confirmed directly to those who return the application blank below and it should be received not later than July 25, 1960

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225 W. Colfax Ave.
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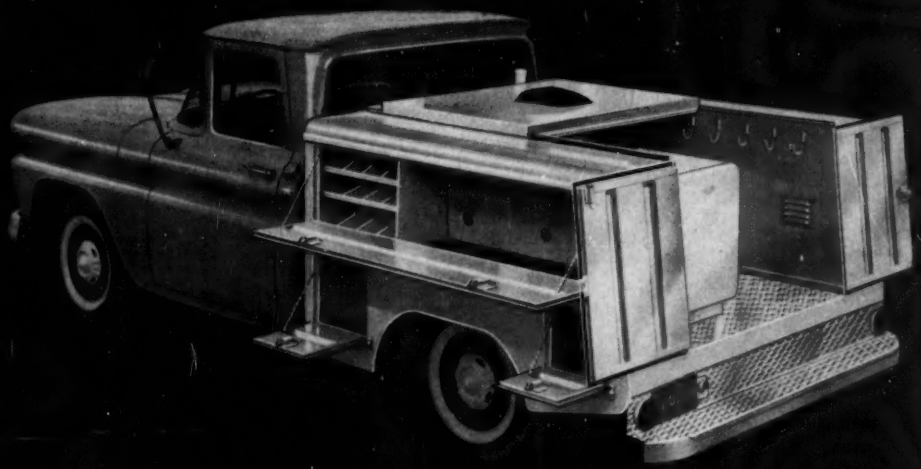

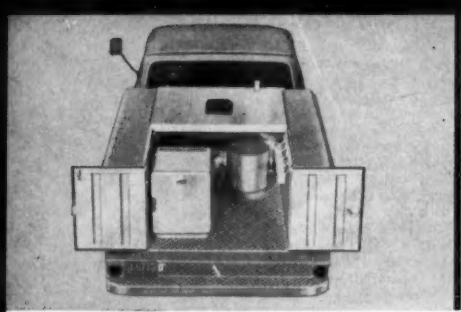

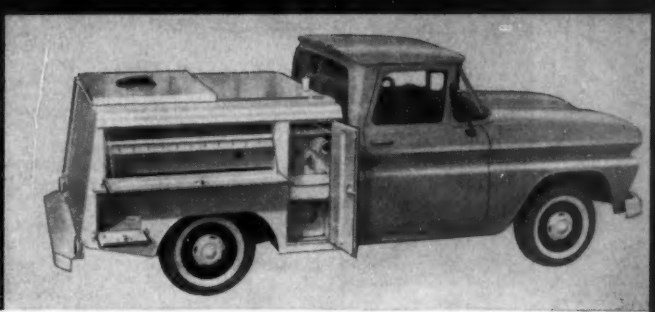

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Coming Meetings

Notices of coming meetings must be received 30 days before date of publication.

Louisiana Veterinary Medical Association, Inc. Annual meeting. Monteleone Hotel, New Orleans, Aug. 1-2, 1960. Robert K. Morris, 406 W. McNeese St., Lake Charles, La., secretary.

American Society of Veterinary Physiologists and Pharmacologists. Annual meeting. Department of Physiology, College of Veterinary Medicine, Colorado State University, Fort Collins, Colo., Aug. 11-12, 1960. The sessions will be held on both days from 9 a. m. until noon and from 1 p.m. until 4:30 p.m. N.H. Booth, Colorado State University, Fort Collins, Colo., president.

American Association of Veterinary Bacteriologists. Annual meeting. Division of Veterinary Science, University of Wyoming, Laramie, Wyo., Aug. 13, 1960. Charles H. Cunningham, Department of Microbiology and Public Health, Michigan State University, East Lansing, Mich., secretary.

American Veterinary Medical Association. Ninety-seventh annual meeting. Denver-Hilton Hotel, Denver, Colo., Aug. 15-18, 1960. H. E. Kingman, Jr., 600 S. Michigan Ave., Chicago 5, Ill., executive secretary.

American Humane Association. Annual convention. La-Salle Hotel, Chicago, Ill., Sept. 26-28, 1960. Mr. R. T. Phillips, 896 Pennsylvania St., Denver 3, Colo., executive director.

National Association of Artificial Breeders. Thirteenth annual convention. Brown Hotel, Louisville, Ky., Aug. 21-24, 1960. Dr. H. A. Herman, 10 North Ninth St., Columbia, Mo., executive secretary.

Central Indiana Veterinary Medical Association. Annual small animal seminar. Claypool Hotel, Indianapolis, Ind., Sept. 14, 1960. P. T. Parker, 5901 Crawfordsville Rd., Speedway 24, Ind., chairman.

New York State Veterinary Medical Society. Sixty-ninth annual meeting. Hotel Syracuse, Syracuse, Sept. 14-16, 1960. Joan S. Halat, New York State Veterinary Medical Society, 803 Varick Street, Utica, N. Y., assistant executive secretary.

Armed Forces Institute of Pathology. Seventh annual course. Armed Forces Institute of Pathology, Washington, D.C., Sept. 26-30, 1960. Deadline for applications is August 15. To apply, write: The Director, Armed Forces Institute of Pathology. Washington 25, D.C.

Helminthological Society of Washington. Fiftieth anniversary. Scientific program will be conducted at the University of Maryland, College Park, Md., Oct. 8, 1960. Helminthological Society of Washington, Animal Disease and Parasite Research Branch, ARS, USDA, Beltsville, Md., publicity committee.

Gaines Dog Research Center. Tenth annual symposium. Kankakee Civic Auditorium, Kankakee, Ill., Oct. 12, 1960. Dean C. A. Brandly, School of Veterinary Medicine, University of Illinois, Urbana, Ill., chairman.

Purdue University. Forty-eighth annual conference for veterinarians. School of Veterinary Science and Medicine, Purdue University, Lafayette, Ind., Oct. 12-14, 1960. Erskine V. Morse, dean.

Eastern Iowa Veterinary Association, Inc. Forty-seventh annual meeting. Hotel Montrose, Cedar Rapids, Oct. 13-14, 1960. Charles B. Thayer, Medical Laboratory Center, S. U. I., Iowa City, Iowa, secretary.

Continued on adv. p. 62

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New England Veterinary Medical Association. Twenty-sixth annual meeting. Sheraton Biltmore Hotel, Providence, R.I., Oct. 16-19, 1960. L. T. Maloney, New England V.M.A. Consultant, 6 Beacon St., Boston, Mass.

United States Livestock Sanitary Association. Sixty-fourth annual meeting. Daniel Boone Hotel, Charleston, W. Va., Oct. 19-21, 1960. R. A. Hendershott, 33 Oak Lane, Trenton 8, N.J., secretary.

Southern Veterinary Medical Association, Inc. Annual meeting. Francis Marion Hotel, Charleston, S.C., Oct. 23-26, 1960. Otto M. Strock, 461 Maybank Highway, Charleston, S.C., general chairman.

Animal Care Panel. Annual convention. Sheraton-Jefferson Hotel, St. Louis, Mo., Oct. 26-28, 1960. Herbert Graff, 835 S. 8th St., St. Louis, Mo., convention secretary.

Cornell University. Annual nutrition conference for feed manufacturers. Statler Hilton Hotel, Buffalo, N.Y., Nov. 2-4, 1960. For programs, preregistration and hotel reservation cards, contact: Prof. Harold H. Williams, Savage Hall, Cornell University, Ithaca, N.Y., chairman.

Missouri, University of. Thirty-sixth annual veterinary conference. University of Missouri, School of Veterinary Medicine, Columbia, Mo., Nov. 7-8, 1960. Cecil Elder, School of Veterinary Medicine, Veterinary Pathology, University of Missouri, chairman.

Arizona Veterinary Medical Association. Annual meeting. Safari Hotel, Scottsdale, Ariz., Nov. 13-15, 1960. Elmer B. Powell, 1102 S. Scottsdale Rd., Scottsdale, Ariz., local arrangement (phone—WH 5-6479).

Wisconsin Veterinary Medical Association. Forty-fifth annual meeting. Schroeder Hotel, Milwaukee, Wis., Jan. 15-17, 1961. W. J. O'Rourke, 540 W. Washington Ave., Madison 3, Wis., secretary.

Arkansas Veterinary Medical Association. Annual meeting. Hotel Marion, Little Rock, Jan. 22-24, 1961. Thayer D. Hendrickson, 7824 Cantrell Rd., Little Rock, Ark., secretary-treasurer.

Minnesota Veterinary Medical Association. Annual meeting. Leamington Hotel, Minneapolis, Minn., Jan. 23-25, 1961. B. S. Pomeroy, 1443 Raymond Ave., St. Paul 8, Minn., secretary.

Ohio State Veterinary Medical Association. Annual meeting. Commodore Perry Hotel, Toledo, Ohio, Feb. 5-8, 1961. Dr. R. E. Rebrassier, 1411 West Third Ave., Columbus 12, Ohio, executive secretary.

Foreign Meetings on adv. p. 64.

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Foreign Meetings

Second International Course on Lyophilization. Lyon, France, Aug. 29—Sept. 9, 1960. For full details, contact: Dr. Louis R. Rey, Directeur des Cours Internationaux de Lyophilisation, Laboratoire de Physiologie, Ecole Normale Superieure 24, rue Lhomond, Paris 5, France.

Fourth International Congress on Animal Reproduction. The Hague, Netherlands, June 5-9, 1961. For additional information contact: the Secretariat of the Fourth International Congress on Animal Reproduction, 14, Burgemeester de Monchyplein, The Hague, Netherlands, Dr. L. Hoedemaker, secretary to the organizing committee.

Eighth International Congress of Animal Husbandry. Hamburg, Germany, June 13, 1961.

Six-Year Course for M.D. Degree Devised

A pilot project, cutting medical training of selected students from the customary 7 or 8 years to 6, has been announced by Northwestern University Medical School, Evanston, Ill. The project will eliminate the sharp dividing line between premedical and medical education, introduce courses that have a direct bearing on the study of medicine in the first years of college, and bring the humanities into medical school.

The pilot group will be made up of 25 students who will go directly from high school into medical school in the fall of 1961. During the first 2 years of liberal arts work, premedical courses will be given in a concentrated form.

Among the main reasons for the program is the hope that the reduced length of study will attract scientifically oriented students who now are discouraged by the long years of medical training. The shorter program also may increase the interest of these students in a combined program for the M.D. and Ph.D. degrees and thus provide individuals needed for careers in academic medicine.

At the end of 4 years under the new curriculum, the students will receive a B.S. degree in one of the biological sciences.—*Mod. Med., 28, (May 15, 1960): 224.*

Horse with Heart Disease Fails in Race

A race horse named Hamama, bought from France for a high price, failed in a race after only 3 furlongs and was walked in "swaying like a drunken man." Immediate and subsequent veterinary examinations indicated a dilated heart. On necropsy 6 months later, fenestrations in the heart were found. No veterinary certificate had been supplied at the time of purchase, although a verbal assurance of soundness was given.

The French owners, as plaintiff, sought payment of about $18,000 which had been withheld after the horse broke down in the race. Judgment was entered in favor of the purchaser.—*Vet. Rec., 71, (1959): 481 (abstr. 1170 in Vet. Bull., April, 1960).*

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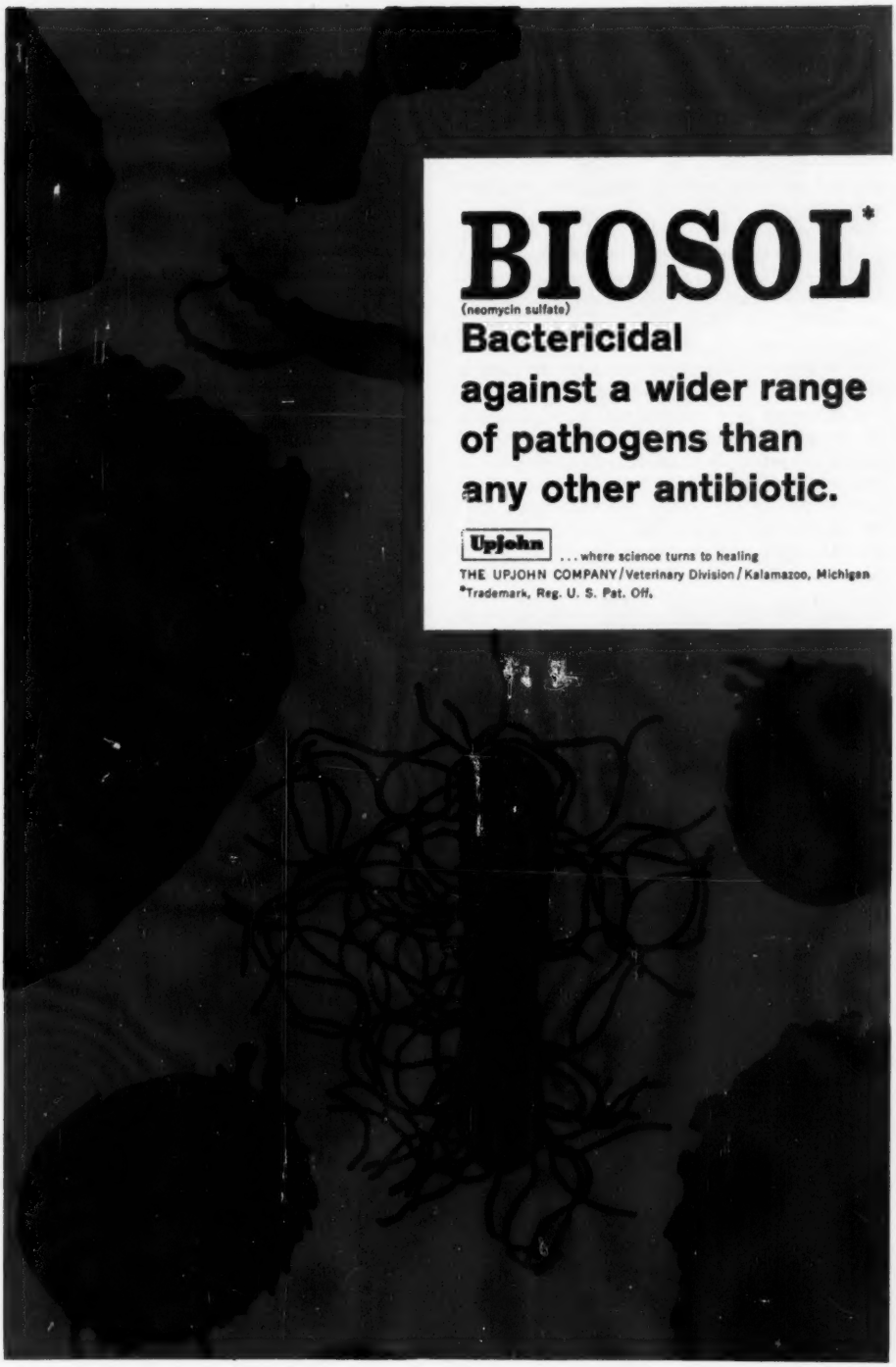
BIOSOL*
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Bactericidal
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any other antibiotic.
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... where science turns to healing
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Beef Tenderized with Pre-Slaughter Injection of Enzymes

A method of tenderizing beef in which the action of the meat's own enzymes is supplemented by an injection of natural food enzymes into the vascular systems of cattle 2 minutes prior to slaughter has been developed by Swift and Company. The enzymes have been widely used in the food field for many years and are not considered additives. The tenderizing potenital is realized when the damped tendering power infused in the meat is released as cooking begins to raise its temperature above 120 F. The tendering power is released in such a manner that a dry-roasted low-choice chuck cut becomes the equivalent in tenderness, flavor, and edibility of a roasted or broiled choice to prime loin or rib. The enzyme is called "ProTen," but it will be some time before marketing tests are complete and the enzyme-tenderized beef will be nationally available.—*Nat. Provisioner, 142, (May 14, 1960): 14.*

Illegal Kangaroo Meat

Evidence recently uncovered indicates that kangaroo meat is being imported from Australia and sold here without disclosure of its source. One firm in Pennsylvania reportedly imported 2 million pounds of meat. The kangaroos are killed in the field, transported under inadequate refrigeration, and marketed here without having received veterinary inspection. They are not generally used for food in Australia today.—*Pennsylvania Dept. of Agric. Weekly News Bull., Jan. 28, 1960.*

CLASSIFIED ADVERTISEMENTS

PERSONAL WANT ADS—$4.00 for the first 25 words and 10 cents for each additional word; 35 cents for use of box number.

TOTAL WORD COUNT must include complete box number address (7 words) or personal address line.

COMMERCIAL WANT ADS—$5.00 for the first 25 words, 25 cents for each additional word; $1.00 for use of box number. (See paragraph above for total word count.)

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DEADLINES

1st of month issue — 8th of month preceding date of issue.

15th of month issue — 22nd of month preceding date of issue.

Names of classified advertisers using key letters can not be supplied. Address your reply to the box number, c/o JOURNAL of the AVMA, 600 S. Michigan Ave., Chicago 5, Ill., and it will be sent to the advertiser.

Wanted—Veterinarians

Wanted—experienced relief veterinarian with New York license for small animal hospital 3-5 weeks in October, November. Westchester County. Sleep in, home-hospital combined. Address Box H 5, JOURNAL of the AVMA.

Wanted—associate veterinarian for mixed practice in Central Oklahoma. State qualifications and salary requirements. Address Box G 56, JOURNAL of the AVMA.

One of the world's largest Greyhound racing organizations requires the services of a well-experienced graduate in veterinary science to assume charge of the medical welfare of several hundred racing dogs. Duties, among other things, would include laboratory work, care of Greyhounds with "track injuries," sprains, fractures, etc., collaborating with trainers in nutritional psychology, conditioning, etc. Organization headquarters in Miami, Fla., with breeding farm in Hendersonville, N. C. Man selected would spend about 8 to 9 months in Miami and balance of time at breeding farm. Please forward resume and salary requirements to Mr. L. L. Crosby, 3567 Bigelow Blvd., Pittsburgh 13, Pa.

Wanted—veterinarian for small animal hospital. Can lease practice, buy, or work on commission and salary. Auburn Animal Hospital, 3115 Auburn St., Rockford, Ill. Telephone WOodland 8-9721 or WOodland 3-7154.

Wanted—Positions

Relief veterinarian available during August and September. Experienced with small and large animals. References. Licensed in Ohio, Michigan, Indiana. Address 22760 Lake Rd., Cleveland 16, Ohio. ED 1-2664.

Veterinarian seeks position with small animal practitioner in Virginia. Licensed, experienced, available immediately. Address Box H 4, JOURNAL of the AVMA.

Wanted—location for veterinarian (Tex '58) in Southwest. Aggressive, married, completes military obligation in October. Address Box H 6, JOURNAL of the AVMA.

Competent energetic recent graduate desires employment leading to partnership in busy small or mixed practice in Southeast. Military obligation completed in September. Address Box H 2, JOURNAL of the AVMA.

Graduate (COL '58), married, fulfills service obligation in September, desires position with work and future, mixed or small animals. Licensed in Colorado, California, Kansas, Wyoming. Address Box E 54, JOURNAL of the AVMA.

For Sale or Lease—Practices

For sale—general practice in Eastern Nebraska, established 5 years. Reason for selling—health. Federal fee work and artificial insemination available. Address Dr. H. E. Jenkins, Box 97, Palmyra, Nebraska.

For sale—well-established small animal hospital and 3-bedroom house in East Bay Area, Calif. Gross over $55,000 yearly. $15,000 will handle. Other interests force sale. Write P. O. Box 3432, Hayward, Calif.

For sale—well-established mixed practice netting over $11,000. Fully-equipped animal hospital, small apartment, 2-way radio. Virginia license required. Very reasonable. Address Box H 3, JOURNAL of the AVMA.

Central Texas air-conditioned hospital, 90 per cent small animal. Real estate, instruments, drugs, x-ray. Ready to walk in and operate. $27,500. $7,500 down. 50 cages. Address Box G 53, JOURNAL of the AVMA.

Need small animal hospital listings immediately. Charles E. Doyle, DVM, Medical practice and real estate broker. 4813 N. McArthur, Oklahoma City, Okla.

For sale—small animal practice in Maryland. Price below year's net. Includes all equipment, x-ray and drugs. Long term lease. Address Box G 54, JOURNAL of the AVMA.

For sale—Southern California small animal practice. Nets $10,000. No real estate; good lease. Price —$6,000 with $2,500 down. Address Box F 21, JOURNAL of the AVMA.

To insure prompt delivery, please address replies accurately:

Box . . . . . .

JOURNAL of the AVMA
600 S. Michigan Ave.
Chicago 5, Ill.

For sale—established Central Illinois general practice. Residence, office, equipment, drugs and office furniture. Easy terms. Address Box F 2, JOURNAL of the AVMA.

For sale—established mixed practice in southern Michigan. Beautiful new house with 5-room clinic attached. Real estate value only—$32,500. Address Box G 4, JOURNAL of the AVMA.

For sale—Minnesota dairy practice with growing small animal portion. House, drugs and equipment. 35 miles from the Twin Cities. Reason for selling—school. Address Box F 52, JOURNAL of the AVMA.

For sale—well-equipped small animal practice on Long Island. $10,000. Real estate under lease with option to buy. Address Box H 1, JOURNAL of the AVMA.

Wanted—Practices

Need small animal hospital listings immediately. Charles E. Doyle, DVM, Medical practice and real estate broker. 4813 N. McArthur, Oklahoma City, Okla.

Miscellaneous

For sale—galvanized cages. 1 9-unit medium size, 1 6-unit large size. Jen-Sal list $560. Operating table. Jen-Sal list $175. Address Dr. Kerns, 2950 N. Hackett, Milwaukee, Wis.

Index to Advertisers

Food Poisoning **Viral Infection**
Bacterial Infection **Malnutrition**

In every case of

DIARRHEA

you'll want

JENOTONE

✓ **Quickly controls diarrhea!**

What it is!

Jenotone is an entirely new and unique antispasmodic which acts directly on all smooth muscle tissue, *including sphincters*, in the respiratory, digestive, urinary and reproductive tracts. Chemically Jenotone is [bis (dimethylamino)-2',3' propyl] - 10 phenothiazine (as the fumarate).

What it does in diarrhea!

Direct, musculotropic action relieves gut spasms without constricting sphincters. Restores *normal* tone to the intestine.

What it will not do!

Will not exert antibacterial or antiviral activity . . . adjunctive therapy sometimes needed.

reliable Jenotone's action is consistent and reliable, when used to relieve intestinal spasms.

safe Jenotone is safe. Acute and chronic toxicity tests show no serious side effects from the normal dosage regimen.

✓ **Eliminates post-treatment constipation!**

effective Jenotone "normalizes" all smooth muscle tissue, including sphincters. Thus it effectively relieves spasmodic conditions associated with a broad range of gastro-intestinal, respiratory and genito-urinary disturbances.

convenient Jenotone therapy is economical and the convenient dosage forms (both peroral and parenteral) make it ideal for dispensing as well as for hospital use.

START WITH A MONTH'S SUPPLY

. . . for a full treatment for a 25 lb. dog based on 2 injections of 2 cc. each and 8 tablets as follow-up treatment.

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15 - 20	#2 bottles	3 bottles	$17.55
20 - 30	2 bottles	4 bottles	$20.30

*Jenotone is also available in 10 cc. bottles.

#Amount shown is approximately one-third more than actually needed.

Write for case reports

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